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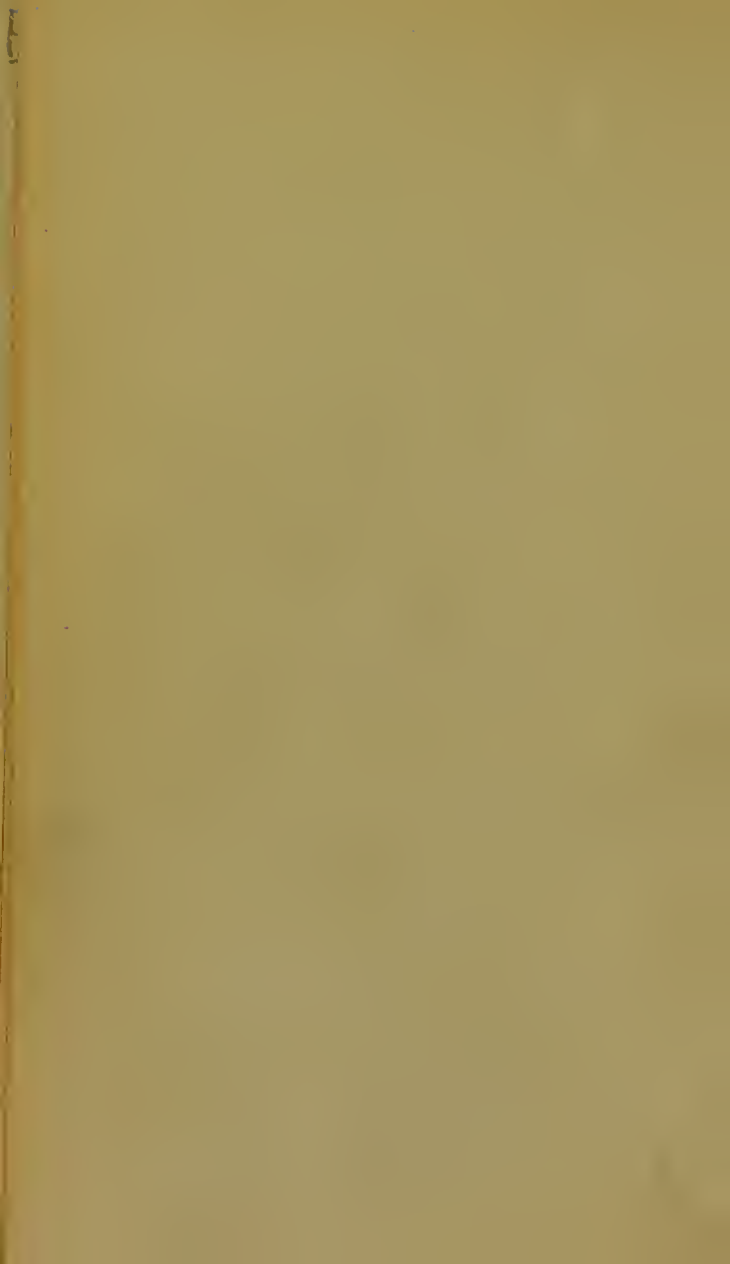
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AIDS TO MEDICINE.

PART I.

GENERAL PATHOLOGY. THE SPECIFIC AND
GENERAL DISEASES.

BY

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PREFACE.

ANY attempt to include the essentials of a large subject within the compass of a small book necessitates the omission of many facts which seem important to the writer, and will doubtless be missed by the reader. Nevertheless, it is hoped that there is enough matter arranged in a convenient form in this book to make it a real 'Aid' to all those who are studying medicine.

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June, 1896.

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AIDS TO MEDICINE.

THE CAUSES OF DISEASE.

ÆTIOLOGY is the name given to the study of the causes of disease. The causes themselves are divided into two classes: (1) the *exciting* causes, which immediately call the disease into being; and (2) the *predisposing* causes, which render the body liable to the development of the disease. For instance, the pneumococcus is the exciting cause of croupous pneumonia, and exposure to cold is the predisposing cause, *i.e.*, the cold lowers the resisting power of the lung, and so enables the pneumococcus to establish itself in it and produce the disease.

The exciting and predisposing causes of disease may be (1) congenital defects, (2) improper use of the organs, (3) harmful influences which act on the body from without.

1. **Congenital Defects.**—There may be actual disease present at the time of birth, such as syphilis, or malformations of the valves of the heart, or a cystic kidney, etc.; but it is chiefly the predisposing causes of disease which are inherited.

For instance, children may be born with a natural weakness of the organs of digestion and assimilation, so that these organs begin to perform their functions imperfectly at a comparatively early age.

In this way indigestion, gout, and other diseases, may occur without any indiscretions of diet being committed by the patient. Also, the children of parents who suffer from such nervous affections as epilepsy, insanity, etc., are born with unstable nervous systems, and are very likely to develop the same diseases.

In some cases of inherited disease, the disease tends to skip a generation; *i.e.*, the grandchildren of the original sufferer are affected, and not his children. This is called *atavism*.

Lastly, there is reason to believe that certain individuals are born with some peculiarity of their blood and tissues which makes their bodies a suitable soil for the growth of disease-germs, so that they readily contract and succumb to the bacterial diseases.

2. The Improper Use of the Organs.—Supposing a child to be born with normal organs, he may acquire disease by the misuse of those organs. Overfeeding may wear out the digestive organs, and lead to indigestion at an early age. Excessive brain-work may produce nervous disorders, and prolonged and arduous muscular work may produce disease of the heart and arteries.

3. Harmful Influences from Without.—The simplest of these are *injuries*, such as blows, wounds, burns, etc. *Cold*, if not an exciting, is certainly a predisposing cause of many diseases, *e.g.*, nasal and enteric catarrh, etc; and excessive cold causes frostbite, or even gangrene. Excessive *heat*, on the other hand, causes sunstroke. Excessive *moisture* of the soil and atmosphere are indirect causes of disease, but certainly favour the development of rheumatism.

As regards *soil*, it must be remembered that certain pathogenic bacteria, such as typhoid and cholera bacilli, may remain alive on the ground, and subsequently soak through into wells containing drinking water. The malarial protozoon also lives in the soil. Further, inorganic materials in the soil may cause disease; for instance, it is thought that goitre may be due to the consumption of water which has filtered through, and is charged with, magnesian limestone.

Occupation may cause disease, either from over-use of a part, as in housemaid's knee, or from the entrance of poison into the body, as in painter's colic. *Mental emotion* may cause functional affections, such as polyuria, and is even thought to be a cause of jaundice. *Improper food*, and, in particular, alcoholic excess, may be classed with the poisons.

Poisons are fruitful causes of disease, and they may either be introduced into the body from without or they may be manufactured inside the body. To the former class belong the common poisons, such as arsenic, opium, etc., which do not concern us here. To the latter class belongs the poison which causes uræmia, and also such substances as carbonic acid and uric acid, when they are in excess in the blood; but it must be noted that this latter class of poisons does not cause primary diseases, but only diseases which are secondary to other affections, *e.g.*, the uræmia to kidney disease, carbonic acid poisoning to lung disease, etc.

Lastly, the bacterial toxins belong to both classes of poisons, for they may be, as will be seen, introduced into the body from without, or may be manufactured by the bacteria inside the body.

PATHOGENIC MICRO-ORGANISMS.

Micro-organisms which cause disease are called *pathogenic*, and they may be animal or vegetable.

The *animal pathogenic micro-organisms* are few in number, as far as we know at present. The two which are best known are the *Plasmodium*, which causes malaria, and the *Amœba coli*, which causes dysentery. The latter can be cultivated outside the human body in hay infusion.

The *vegetable pathogenic micro-organisms* chiefly belong to a class of fungi, called the *schizomycetes*, or *fission fungi*, because they multiply by elongating and dividing into two. Some of them, however, form spores, from which new ones may develop. They are often called collectively *bacteria*, and they are classified according to their shape, as follows :

If they are rod-shaped, they are called *bacilli*. Anthrax, tuberculosis, leprosy, tetanus, diphtheria, etc., are caused by bacilli.

If they are globular, they are called *micrococci*. Septicæmia, erysipelas, gonorrhœa, pneumonia, etc., are caused by micrococci. Cocci are also named according to their methods of grouping : for instance, if they occur in pairs, they are called *diplococci*, *c.g.*, the pneumococcus ; if they occur in chains, like a string of beads, they are called *streptococci*, *e.g.*, the cocci of septicæmia and erysipelas ; and if they occur in more or less rounded groups they are called *staphylococci*, *c.g.*, the micrococci which are the cause of various suppurative diseases. The gonococci often occur in groups of four.

If bacteria are twisted, like a corkscrew, they are called *spirilla*. Relapsing fever is caused by

a spirillum, and it is probable that the so-called cholera bacillus is really a broken spirillum.

Bacteria can be stained with various preparations of the aniline dyes, and most of them can be cultivated outside of the body, *i.e.*, they grow readily in such materials as potatoes, blood serum, jellies made of gelatine and peptone, or agar-agar and peptone, etc. These materials are called *nutrient media*. A pure cultivation is one in which only one species of bacterium is growing; and it has been found that different kinds of bacteria produce different appearances in the nutrient media in which they grow, so that cultivation is a good means of distinguishing one micro-organism from another.

In order to prove that a particular bacterium is the cause of a particular disease, it is necessary for the organisms to fulfil certain conditions which are called *Koch's laws*. These are:

1. The organism must invariably be found in the blood, tissues, or excretions of a person suffering from the disease.

2. The organism must be shown to differ from all other organisms. The difference may be in its size or shape, its method of grouping, its staining reactions, and its method of growth when cultivated.

3. The organism must be separated from all organic or inorganic particles which came with it from the body of the patient. If this were not done, the result of future inoculations of the organism would be inconclusive, because the effects produced might be due to other substances than the organism. The separation is effected by cultivating the organism out of the body, and if more than one organism be present, it is easy to distinguish one from the other, and to inoculate fresh media

with the one selected for experiment, until a pure cultivation is obtained.

4. Lastly, a healthy animal must be inoculated with a pure cultivation of the organism, and must become affected with the original disease. The organism must also, of course, be found in the blood, etc., of the new animal.

Bacteria produce disease by manufacturing poisons, which are called *toxins*, out of the fluids and tissues of the body in which they grow, and they make much the same toxins out of the nutrient media in which they are cultivated. Chemically the toxins are either *ptomaines* or *albumoses*.

Now, it has been proved that the symptoms may be very different, according to whether the bacteria themselves or only their toxins enter the body. In the latter case the symptoms will be proportional to the amount of poison which obtains entrance, because the poison cannot multiply. But in the first case no such proportion exists, because even if only a few bacteria enter the body, they multiply rapidly to thousands, and so the manufacture of toxins goes on increasing enormously. In other cases there is a qualitative difference in the symptoms; for instance, if the diphtheritic bacilli be inoculated on to a mucous membrane, a false membrane is produced, but if the toxins only be introduced there is no membrane formed, but the other symptoms of diphtheria, such as albuminuria and paralysis, follow in due course.

Experimentally, the toxins can be obtained without the bacilli by boiling the cultivations until the bacilli are dead, when the toxins can be used for inoculations. A practical instance of pure toxic effects is sometimes afforded by a putrefying clot

inside the uterus. The bacteria grow in the clot and produce their toxins there, and the toxins may be absorbed into the blood without any bacteria accompanying them. In such a case rapid recovery takes place when the uterus is washed out ; but if the bacteria enter the blood, as they tend to do eventually, the condition becomes much more serious (see Septicæmia).

When bacteria have been producing toxins in the body for some time, many of them start producing a new substance, which neutralizes the toxins, and may kill the bacteria themselves. A kind of self-poisoning occurs, and it is by this process that many specific diseases come to an end of their own accord, after a certain number of days, if the patient lives long enough. The new substance is called the *antitoxin*, and its production brings the disease to a close. Antitoxins can, in some cases, be manufactured by cultivating bacteria in special ways, and they may be extracted from the blood serum of animals to which a disease has been communicated in a modified form, and such antitoxins, if injected into a human being with the disease, may cure him. The antitoxins of tetanus and of diphtheria are obtained by these methods.

Vaccines are substances which, when inoculated, produce a disease in a mild form, or even without any perceptible symptoms. But the effect is that the individual inoculated, having had this mild form, is protected from taking that disease in the usual way for a considerable period, and perhaps for ever ; or if he be not absolutely protected, and so does contract the disease, he has it in a mild form. Some antitoxins, such as that of diphtheria, are said to be vaccines as well as antitoxins.

Vaccines can be prepared by cultivating bacteria in special ways. Also it has been found that the blood of certain animals has the power of modifying the virulence of certain bacteria. Hence, if these animals be inoculated with these bacteria, they take the disease in a mild form, and then vaccines against the disease can be obtained from their blood or tissues, and can be used to protect human beings.

Vaccines are in use for small-pox and cholera.

THE SYMPTOMS OF DISEASE—CASE-TAKING.

It is usual to divide the clinical phenomena of disease into *symptoms* and *physical signs*, the latter being the phenomena which are detected by examining the patient. In order to study the symptoms of disease, a systematic method of case-taking must be adopted, to which end the following hints may be useful:

Put down first the *name* of the patient, and the *symptoms of which he complains*.

Thus, take his *personal history*, past and present. This includes:

(a) His *age*, which is often important. For instance, jaundice in an old person is probably due to cancer, but in a young person to catarrh of the bile-ducts.

(b) His *social state*, whether married or single.

(c) His *occupation*, remembering that painters, miners, etc., are liable to special diseases.

(d) His *habits*, with especial reference to intemperance, over-eating, and excessive smoking; to excessive or deficient exercise, sleeping, etc.

(e) His *residence*, past and present, which is important, because certain diseases, such as malaria,

etc., are endemic, *i.e.*, they are always to be met with in certain localities.

(f) Any *diseases from which he may have suffered*. The importance of rheumatic fever as a cause of heart disease, of scarlet fever as a cause of kidney disease, etc., cannot be overestimated.

Then take his *family history*, because many diseases, such as tuberculosis, gout, etc., are hereditary. In some cases, such as hæmatophilia, etc., it is necessary to inquire about the grandparents and great-uncles, especially on the maternal side.

The next thing to do is to take the *history of the present attack*, carefully noting the sequence of the earliest symptoms, the surroundings of the patient at the time, and inquiring for any events which may have preceded the symptoms. For instance, chorea may come on a day or two after a fright, pneumonia shortly after a wetting, etc.

Lastly, it is necessary to examine the patient and note his *present condition*, carefully comparing what is observed with what he has complained of. For instance, if he complained of cough, and the lungs prove to be normal, the cough is probably laryngeal or reflex.

In examining the patient, the various organs, the heart, pericardium, arteries and blood, the lungs and pleuræ, the alimentary canal from the lips to the anus, the liver, pancreas and kidneys, the spleen and lymphatic glands, the thyroid and suprarenal, the brain, cord, muscles, nerves, and special sense organs, and the sexual organs, should be systematically inquired after and examined. Details of these examinations are given under the diseases of the various organs.

In addition, it may be noted that the *facies* may

indicate pain or exhaustion, and that the pallor of anæmia, the cyanosis of heart disease, the dropsy of kidney disease, the alterations of the pupil of paralysis, and many other points, may be revealed by inspecting the face.

The *decubitus*, or manner of lying down, may be noteworthy. In the prostration of fever, patients lie on the back, and in other affections they may lie on either side to spare a tender part, or, in the case of lung disease, to give the sound lung free scope to expand. In severe dyspnœa they sit up.

If a patient can stand, his method of standing and of walking may be quite characteristic of certain diseases.

The *nutrition of the body* should always be observed, and in many cases it is advisable to weigh the patient once a week.

The *colour of the skin* is important in jaundice, Addison's disease, etc., and the presence on it of eruptions or of scars denoting syphilis may be a great help to diagnosis.

The *temperature of the body* should be 98·4 F., and any temperature above that indicates fever. Hyperpyrexia occurs sometimes in rheumatic fever, and even in the other fevers, and subnormal temperatures are met with after a long fever, and in jaundice, cholera, and other cases of collapse, etc.

HYPERÆMIA—CONGESTION.

These terms are synonymous, and they mean that *the capillaries are distended with blood*. There are three varieties of congestion—active, passive, and mechanical.

1. **Active or Arterial Congestion.**—In this case the arteries become dilated (usually through paralysis of the sympathetic nerves which supply them), and consequently an excess of blood reaches the capillaries and distends them. This kind of congestion is usually local, *i.e.*, confined to one part of the body. It occurs in the face during blushing, in lesions of the sympathetic ganglia in the neck, in bad cases of neuralgia of the fifth nerve, in some cases of exophthalmic goitre, etc. It is supposed to occur in the liver after over-eating, and in diabetes. But most commonly active congestion, especially in the lungs, is only a prelude to inflammation. In active congestion the affected parts are red and hot.

2. **Passive Congestion.**—In this case the capillaries become distended on account of a lax condition of their walls. The results are much the same as those of mechanical congestion.

3. **Mechanical or Venous Congestion.**—In this case the veins are obstructed, so that the blood does not escape from the capillaries, and therefore distends them. The condition may be *general* or *local*.

General mechanical congestion occurs in heart disease, and sometimes in lung disease, because the blood becomes dammed up in both *venæ cavæ*, and consequently the capillaries all over the body become distended.

Local mechanical congestion may occur in the portal system, from obstruction to the vein in cirrhosis or cancer of the liver, or in the leg from blocking of the femoral vein by a thrombus, and in many other parts of the body.

Parts suffering from mechanical congestion are

cold, blue, and a little swollen. If œdema ensues, as it often does, the swelling may be very great.

Mechanical congestion, when general or portal, may be treated by purgatives and diuretics, so as to remove a certain quantity of water from the capillaries, and if the dropsy be great, tapping may be performed. If it be confined to the leg, the recumbent position should be maintained as much as possible, and an elastic stocking should be worn.

DROPSY—ŒDEMA.

Dropsy is not a disease in itself, but a pathological condition which is secondary to some disease. The process in dropsy consists in the exudation of *serum* through the capillary walls into the connective tissue or into the serous cavities. In the former case the serum separates the connective tissue fibres and produces a soft swelling, which *pits on pressure, i.e.*, if compressed for a few seconds the serum is displaced, and when the pressure is taken off it does not return immediately to the compressed part, so that a pit or hollow is left. In the serous cavities the serum may accumulate, so that a large bag of fluid is produced, which may compress the adjacent organs. Serum is, of course, highly albuminous, and contains salts and extractives. It is neutral in reaction, and the specific gravity varies from 1003 to 1016.

Dropsy of practically the whole subcutaneous connective tissue is called *anasarca*, and in *general anasarca* dropsy of the serous cavities is included. Dropsy of the connective tissue of a localized part, such as the legs or lungs, is called *œdema* of that part, while dropsy of the serous membranes has a

special name in each case. Thus, dropsy of the peritoneum is called ascites; of the cerebral ventricles, hydrocephalus; of the pleura, hydrothorax; of the pericardium, hydropericardium; and of the tunica vaginalis, hydrocele.

General dropsy is most often met with in heart disease, in kidney disease, and in anæmia.

In cardiac dropsy the blood becomes dammed up in the veins because it cannot get through the heart. This leads to mechanical congestion of the capillaries, and stagnation of the blood in them. Now, as stagnant blood is always loaded with carbonic acid and other effete substances absorbed from the tissues, the capillary walls become impaired, and allow the serum to pass through them. In some cases of pulmonary disease, general dropsy may be brought about in the same way.

Cardiac dropsy begins in the feet, and gradually ascends.

In renal dropsy the blood is deficient in albumen, because so much albumen is passed in the urine. Consequently the capillary walls are ill-nourished, and allow the serum to pass through. Another factor in causing renal dropsy is the diminished elimination of water by the kidney. Renal dropsy begins in the eyelids and scrotum, and tends to become universal, the serous cavities being rapidly filled up.

In anæmia the impoverished condition of the blood is the chief cause of the dropsy, but the weakness of the heart may have a share in producing it. It is most marked in the feet.

In localized dropsy, whether of the connective tissue or serous cavities, the cause is nearly always mechanical congestion. Thus, the pressure of a

tumour on the common iliac veins causes œdema of the legs; blocking of the portal vein by cancer of the liver causes ascites; and compression of the *venæ Galeni* causes hydrocephalus, etc. But in these cases the mechanical congestion acts, as in cardiac dropsy, by affecting the nutrition of the capillary walls. It is usual to class the large accumulations of serum which occur in chronically-inflamed serous cavities with dropsy of those cavities. For instance, if in chronic peritonitis much fluid be exuded into the peritoneum, the condition is called ascites.

Dropsical organs are particularly liable to become inflamed, and, especially, to be infected by septic micro-organisms.

The treatment of dropsy depends upon its cause, purgatives being useful in most cases, digitalis and diuretics in heart disease especially, and iron in both renal and anæmic cases.

LARDACEOUS DISEASE.

This is, like dropsy, a secondary affection. The diseases which lead to it are: (1) any disease in which suppuration goes on for a long time, such as tubercular disease of the vertebræ or other bones, phthisis, chronic pyelitis, etc.; (2) syphilis, even without suppuration. It is also met with in chronic nephritis.

The process in lardaceous disease consists in the deposition of a new material called lardacein in the walls of the arterioles and capillaries and in the connective tissue. This material must leave the blood in a fluid condition, but when deposited in the tissues it becomes solid.

Lardacein is chemically an albuminoid body, so the disease is often called *albuminoid* disease. Formerly it was thought to be a starchy body, which accounts for its old name of *amyloid* disease. It is allied to fibrin in its chemical characters, and it has been suggested that lardacein is really dealkalinized fibrin, and that the loss of alkali which the blood suffers during suppuration accounts for its production and subsequent transudation.

To the naked eye lardacein is firm, gray, and translucent. It is stained a deep-brown colour by a solution of iodine in iodide of potash and water, while the normal parts are stained yellow. If sections of a lardaceous organ be stained with methyl violet, the lardaceous parts come out lake-red in colour, and the normal parts a blue violet. This colouration is very conspicuous under the microscope, and the red lardacein has a structureless, translucent, or misty appearance.

Lardaceous infiltration may affect all parts of the body, but it is usually most marked in the liver, spleen, kidney, and intestine. In all these organs the lardaceous change begins in the walls of the essential vessels of the organ—in the capillaries from the hepatic artery in the liver; in those arterioles of the spleen on which the Malpighian bodies are situated; in the arterioles of the Malpighian bodies of the kidney; and in the vessels of the villi in the small intestine. As it spreads beyond the vascular walls, the lardacein infiltrates the connective tissue of the organ, and destroys the cells by pressure.

Lardaceous organs are larger, heavier, firmer, and paler than normal, and functionally lardacein

appears to increase the permeability of the vascular walls, for in the kidney it leads to polyuria, and in the intestine to diarrhœa.

INFLAMMATION.

Inflammation is the name given to the series of changes which occur in the living tissues and blood, in consequence of the infliction of some injury. The portions of tissue which are destroyed by the injury, being dead, take no part in the inflammatory process. The word *irritation* is used to express the way in which the injury acts on the living tissues.

The causes of inflammation are therefore *irritants*, and they are of two kinds—acute and chronic.

Acute irritants act rapidly and severely. Such are wounds and burns, chemical irritants like arsenic, and certain bacteria and their toxins. These irritants cause *acute* inflammation.

It must be noted that if a wound be clean-cut, and not infected by bacteria, it causes very little irritation, because the only source of irritation to the living tissues is the presence of the few cells which have been killed by contact with the knife, and these are not in themselves highly irritating.

Chronic irritants act slowly and slightly, but for a long period. The presence of an excess of uric acid or alcohol, etc., in the blood causes chronic irritation of the tissues; and syphilis, and many other diseases, act in the same way. Further, any dead material in the body, such as a bullet or a blood-clot, is a source of chronic irritation to the living tissue around it. Such irritants as the above cause *chronic* inflammation.

The Phenomena of Acute Inflammation.

(A.) **The Inflammatory Process**—1. *Changes in the Bloodvessels and Blood*.—(a) There is dilatation of the arteries, capillaries, and veins, which accelerates the blood-stream. Up to this point only acute congestion is present. (b) The blood-stream now becomes slow, and in the centre of the inflamed area it stops altogether. This is called *inflammatory stasis*, and it indicates the transition from acute congestion to inflammation. (c) The white blood corpuscles adhere to the walls of the veins and capillaries.

2. The *Exudation* now occurs. It consists of (a) the white blood corpuscles (leucocytes), which pass through the walls of the veins and capillaries into the tissues around; and (b) a fluid which is like serum, but contains fibrinogen. This fluid may coagulate and deposit fibrin, in which case the inflammation is called *croupous*. But in the presence of certain bacteria, which are called *pyogenic*, no coagulation occurs, and the leucocytes mix with the fluid to form *pus*. If pus be formed in a solid organ, it collects in a cavity which is called an *abscess*.

3. *The Changes in the Cells*.—The irritant may be severe enough to kill a large number of cells. For instance, the *core* of a carbuncle is a collection of cells which have been killed in this way. But if the irritant be less acute, it makes the cells proliferate, *i.e.*, multiply rapidly. In mucous membranes the proliferation of the epithelium may cause an abundant secretion, in which case the inflammation is called *catarrhal*. In connective tissue the proliferated cells may act as phagocytes, or go to form granulation tissue.

(B.) **The Terminations of Acute Inflammation—1.** *The Inflammation may extend indefinitely.*—This especially occurs when septic bacteria are the irritant. The exudation and destruction of cells goes on over a wider and wider area, until the death of the patient is effected.

2. *The Inflammation may be completely arrested.*—To procure this result, the irritant must cease to irritate, and any dead matter produced by the inflammation must be removed or isolated in some way, so that it is no longer a source of irritation.

Now, some irritants, such as burns, cease to irritate spontaneously; others, such as the less virulent bacteria, may be eaten up by leucocytes and proliferated connective tissue corpuscles, so that their action ceases. The devouring of living bacteria or dead particles by living cells is called *phagocytosis*, the cells themselves being called *phagocytes*. If the bacteria be more virulent, the phagocytes attempt to destroy them, but are killed by the toxins, and thrown off as pus-cells, and the irritation does not cease until the toxins are neutralized by some antitoxin (see p. 7).

In any case, when the primary irritation has ceased, the removal of the dead matter is undertaken. Fluids, such as exuded serum and oil from fatty cells, are absorbed by the lymphatics. Solid matter, if small in quantity, is eaten up by phagocytes; but if larger it is removed by *granulation tissue*. This tissue is composed of proliferated connective tissue corpuscles and of new capillaries, which have grown from pre-existing ones. It forms around the dead matter, grows into it, and replaces it, the granulation tissue cells eating up the dead particles. Then the cells become spindle-

shaped cells, and finally fibres, so that the dead matter is replaced by a fibrous scar. If the quantity of dead matter be still greater, or if it be an irremovable substance like a bullet, the granulation tissue grows round it, and then developes to fibrous tissue, so that the dead matter is surrounded by a fibrous capsule, and can no longer irritate the soft tissues.

3. *The Inflammation may become Chronic.*—In this case the strength of the irritant and that of the arresting forces (the phagocytes and antitoxins) is evenly balanced, so that, on the one hand, the inflammation does not extend, and on the other it cannot be completely arrested. This is the state of affairs in chronic abscesses and sinuses, in gleet, and in some cases of bronchitis and nephritis, etc. The inflammatory processes described above go on with moderate intensity, but for a long period, and granulation tissue is very likely to be formed.

Chronic Inflammation.

When the irritation is of a chronic nature from the beginning, the inflammatory process consists in slight hyperæmia and a slow exudation of leucocytes. The old cells of the part die almost one by one, and leave very little débris. On the other hand, the proliferation of cells is a marked feature of chronic inflammation, and in particular there is nearly always an abundant growth of granulation tissue by proliferation of the connective tissue corpuscles, and as this tends to develop into fibrous tissue, a chronically-inflamed organ usually exhibits a great increase of its fibrous tissue.

The Symptoms of Inflammation.—These are: (1) *Pain*, due to injury to, and compression of, the

nerves, particularly if the exudation be pent up beneath an unyielding fascia; (2) *redness*, due to hyperæmia; (3) *local heat*, due to hyperæmia; and (4) *swelling*, due to hyperæmia, swollen cells, and exudation. There is general fever if the toxins enter the blood, or perhaps if there is great local tension.

The Treatment consists in relieving the pain by poultices, etc., in aiding the removal of dead materials by early incision, tapping, etc., and, in special cases, in the use of antitoxins. A calomel purge at the beginning, followed by salines, is often useful.

FEVER—PYREXIA.

The phenomena of fever consist in a rise of temperature, an increase in the rapidity of tissue change, and an alteration in the secretions, which become less in quantity and altered in quality. In detail the symptoms are as follows:

(a) *Rise in Temperature*.—The increased rate of tissue change means increased chemical action, and therefore an increase of *heat-production*. Other sources of increased heat-production are the chemical reactions between the bacteria and their toxins on the one hand, and the fluids and tissues of the body on the other. At the same time, the dryness of the skin and the diminution of the excretions diminish the usual sources of *heat-loss*, so that, as a total result, the body temperature is raised.

In a normal person increased heat-production is immediately compensated for by increased heat-loss by the action of the heat-regulating centres in the brain, and the result is that the temperature is kept at 98·4° F. Now, it is thought that in fevers

these centres are poisoned, so that they do not perform their functions properly. In particular, they do not seem to be able to procure heat-loss, so that heat accumulates in the body; but it is also probable that the poisoning of these centres permits an increase in metabolism, *i.e.*, in the amount of tissue change, and so causes increased heat-production.

A temperature over 105° constitutes *hyperpyrexia*. In children and puerperal women high temperatures may occur from trivial causes, such as indigestion, constipation, a slight chill, emotion, etc.

(b) **The Skin.**—In most fevers the skin is *hot* and *dry*, from diminished secretion of sweat. The exception is rheumatic fever, in which profuse sweating occurs, while the temperature is high. In the other fevers, if sweating does occur, it usually coincides with a drop in the temperature. At the end of a fever sweating is often profuse.

(c) **The Circulatory System.**—The pulse is always *increased in frequency*. In certain cases, called *sthenic fevers*, it is also full, strong, and bounding, while in others, called *asthenic fevers*, it is weak and dicrotic, and sometimes small. As a rule, the increased pulse-rate is proportional to the increased temperature, but in acute tuberculosis it may not be so.

In prolonged or malignant fevers there is passive congestion of the capillaries in dependent parts of the body.

The effect of the fever on the heart is revealed by the pulse, but in bad cases, when exhaustion sets in, there may be reduplication of either the first or second sounds.

In the blood, the red discs are diminished in number (oligocythæmia), and the white may be slightly more numerous (leucocytosis). The fibrin is said to be increased (hyperinosis).

(d) **The Respiratory System.**—The rate of breathing is *increased*, and the expired air contains more carbonic acid. As a rule, the increased rate of breathing is proportional to the increased pulse-rate, but croupous pneumonia is an exception, the breathing being disproportionately increased.

(e) **The Alimentary System.**—There is *thirst*, and the tongue is *dry*, and usually *furred*. Sometimes it is raw and beefy. It may be glazed, and in bad cases it may become dry, brown, and tremulous (typhoid state). There is complete loss of appetite (anorexia). Vomiting in the late stages may be gastric, but in the early stages is cerebral. *Constipation* is the rule, but typhoid fever is a notable exception to this. As a result of the changes in the alimentary system, and of the increased waste of tissue, there is always *emaciation* in fever.

(f) **The Urine.**—This becomes scanty, very acid, high-coloured, and has a high specific gravity. It contains an excess of urea and uric acid, and of the various salts, with the exception of the chlorides, which are diminished. In bad cases nephritis, or degeneration of renal epithelium, may occur, and then there is albuminuria.

(g) **The Nervous System.**—The vomiting and rigor which occur at the beginning of so many fevers are due to the first effect of the toxin on the nervous centres. A rigor is a shivering attack of more or less intensity. In slight cases there may be merely a sensation of cold, but in bad cases actual shuddering may occur. In children there

may be a convulsion instead of a rigor. Headache is a common symptom in fevers, and the symptoms of the *typhoid state* are chiefly nervous symptoms.

The Typhoid State.

This consists in the presence of a certain group of symptoms. These are: (1) a dry, brown, tremulous tongue, with sordes, *i.e.*, brown patches of dried mucus and epithelium, on the lips and teeth; (2) subsultus tendinum, *i.e.*, slight muscular contractions, which move the tendons to a slight extent, and may cause tremors of the hands, etc.; (3) carphology and floccitatio, *i.e.*, picking at the bed-clothes or at specks in the air; (4) mental confusion and drowsiness, which go on to delirium. The delirium is low and muttering in asthenic cases, but in sthenic cases there may be great excitement, and even violence. In alcoholic persons the delirium produced by a fever may have all the characteristics of delirium tremens. Eventually coma sets in, and the urine may be retained at first, but is afterwards passed involuntarily, as are the fæces. At this stage the patient lies, a helpless mass, on the bed, and bedsores are liable to form. (5) The heart and pulse indicate asthenia, the latter being rapid, weak, and dicrotic.

It must be understood that the typhoid state comes on gradually, the tongue gradually getting dry and brown, and the mental confusion gradually going on to delirium and coma. Also, all of the symptoms may not be present. Hence, in practice, a patient is often described as showing signs of getting into the typhoid state, or being somewhat in a typhoid state, or, in the fully-developed cases, being deeply in the typhoid state.

Pathology of the Typhoid State. — It appears certain that the symptoms of the typhoid state are due to a poison circulating in the blood, and particularly to the effects of that poison on the nervous system. The poison consists of the *products of tissue change, which accumulate in the blood, because they cannot be eliminated by the excretions*; but nowadays it is necessary to include among these products the poisons which bacteria make out of the fluids and tissues of the body.

A high temperature appears to assist in the production of the poison, because in most persons, if the temperature remains over 103° without remission for four or five days, some or all of the symptoms of the typhoid state come on. Further, in many cases of fever the typhoid state can be warded off by systematic antipyretic treatment. It would appear that the high temperature causes degeneration of the epithelium of the liver, kidney, etc., and of the muscular fibres and other connective tissues, and that the products of this degeneration are poisonous, and as they are not freely eliminated, they accumulate in the blood.

On the other hand, the typhoid state may occur independently of fever, as, for instance, in acute yellow atrophy of the liver. But in this case there is the same degeneration of epithelium, etc., and the same difficulty in eliminating the products of degeneration as in the fevers, although there is no high temperature. Consequently the same symptoms result.

In practice, the typhoid state is most frequently met with in typhus and typhoid fevers. But it may occur in any fever provided the temperature keeps high long enough to cause degeneration of

the tissues. It also occurs in acute yellow atrophy of the liver, and in phosphorus-poisoning, and sometimes in uræmia and diabetes, in all of which a high temperature does not necessarily occur.

Classification of Fevers.

Fevers may be classified in various ways.

They may be classified according to their effect on the patient's strength as follows :

1. *Sthenic fevers*, in which the pulse is full and strong. Most fevers, *e.g.*, scarlet fever, small-pox, etc., begin sthenically when they attack young persons, but become asthenic after a time. But croupous pneumonia is the best type of a sthenic fever, because the pulse may be full and strong to within a few hours of death.

2. *Asthenic fevers*, in which the pulse is weak and compressible from the first. Diphtheria and many cases of typhoid are examples of this type.

Fevers may also be classified according to the temperature chart as follows :

1. *Continuous fevers*, in which the temperature remains high all day for several days. Typhus and pneumonia are instances of this class.

2. *Remittent fevers*, in which the temperature falls to a definite extent (one degree or more) every twenty-four hours, but without ever reaching the normal. The fall of temperature is called the remission, and it most often occurs in the morning. Examples of this class are certain malarial fevers, typhoid fever (except during the last few days), septicæmia, and the fever of phthisis.

3. *Intermittent fever*, in which the temperature reaches the normal once in every twenty-four hours. Examples of this class are the common malarial

fever, typhoid fever in the last few days, and septicæmia and phthisis occasionally.

4. *Relapsing fever*, in which the temperature keeps high for a few days and then falls and keeps normal for a few days. After this it rises again for a few days, and then is normal for a few days, and so on. The so-called relapsing fever is an example of this, but it may occur in certain cases of suppuration, *e.g.*, hepatic or cerebral abscess.

Fevers used to be classified according to their cause into *specific* or *primary* fevers, in which there was no local lesion, but a general blood-poisoning, *e.g.*, scarlet fever; and into *secondary* or *symptomatic* fevers, in which there was a local inflammatory focus which caused the rise of temperature, *e.g.*, pneumonia, erysipelas, etc. But as most of the latter class of affections have been shown to be specific, the distinction will not entirely hold. But although the term specific no longer distinguishes one class from another, still the high temperature may be due to two very different pathological conditions. For instance, in diphtheria and erysipelas the poison which causes the fever is absorbed from a local lesion, the fever is proportional to the amount of poison absorbed, and when the local lesion gets well, the fever ceases. On the other hand, in typhus the poison seems to be in the blood from the first, the fever produced by it runs a definite course, and only ceases when the poison in the blood is exhausted.

THE FEVERS.

General Considerations.—The *incubation period* of a fever begins when the poison enters the body, and

ends when the first symptom occurs. The *period of invasion* is the period during which the early symptoms are occurring. The onset of a fever is said to be *acute*, if the early symptoms come on suddenly and are severe, as in typhus, small-pox, pneumonia, etc. If the symptoms develop slowly, and the early ones appear mild, the onset is *gradual*, as in typhoid.

The following points concerning the *rash* of a fever must always be noted: (1) The day of the fever on which it appears; (2) the part of the body on which it is first seen; (3) its colour; (4) whether it is raised or not; (5) whether it fades on pressure or not; (6) the shape of any spots or patches which occur; (7) any changes which occur in it, *e.g.*, the maturation of the variolous papule; (8) its duration.

Terminations of a Fever.—Certain fevers, *e.g.*, typhus and croupous pneumonia, end by *crisis*, *i.e.*, the temperature drops from perhaps 103° to normal in a few hours, and then keeps normal. With the fall of the temperature *critical discharges*, such as profuse sweating, etc., may take place. Other fevers, *e.g.*, typhoid, end by *lysis*, *i.e.*, the temperature falls slowly, taking several days to reach the normal. The fall of the temperature is called *defervescence*. After a long fever it is usual for the temperature to be subnormal for some days.

Treatment of Fever.

This may be (1) radical, (2) expectant.

The *radical* method of treatment consists in trying to cut short the fever. Formerly this meant that we tried to eliminate the poison by purging and

sweating. Nowadays we try to kill the germ which causes the fever by giving antiseptic drugs, or to neutralize the poison by the use of antitoxins (see p. 7). As regards drugs, quinine stops malarial fever by killing the protozoon, and some authorities think that mercury, if given early in the disease, will kill the typhoid bacillus, and so cure typhoid fever. As regards antitoxins, those of diphtheria and tetanus are in general use, and certainly appear to arrest the diseases whose names they bear.

The *expectant* method of treatment consists in letting the disease run its course, and merely treating any symptoms which are distressing, or which threaten life. For instance, the sore throat of scarlet fever, the headache of typhus, the diarrhœa of typhoid, etc., require treatment, although we cannot arrest the disease itself. Also, the patient's strength must be maintained by good nursing and suitable diet; weakness of the heart must be treated by brandy and strychnia, and the temperature may with advantage be regulated by antipyretic treatment. Lastly, dangerous complications, such as hæmorrhage, pneumonia, etc., require very active measures. In all cases in which consciousness is impaired, the bladder must be carefully watched, as retention is likely to occur.

The *diet* of a fever patient should consist of milk and beef-tea. Chicken-broth, calf's-foot jelly, beaten-up eggs, and other light and easily-digestible articles of diet, may also be allowed. The *feeds* should be given at regular intervals. If the patient can take 3 pints of milk and 1 pint of beef-tea in the twenty-four hours, it may be considered satisfactory. Brandy may be given in proportion

to the cardiac weakness, from 12 to 16 oz. a day being the maximum quantity permissible.

The Antipyretic Treatment of Fever.—This treatment is based on the theory that the mere heat causes degeneration of the tissues, particularly of the cardiac and voluntary muscles, and of the epithelium of the liver, kidney, etc. Such degeneration causes cardiac weakness and deficient excretion; and, further, as the products of degeneration are absorbed into the blood, and are not eliminated freely, they act as poisons, and bring on the typhoid state. Hence, if the temperature can be kept within bounds, even by artificial means, we save the heart and keep off the typhoid state, although we do not cut short the disease. It follows that, to be of real use, the antipyretic treatment must be begun early, before degeneration has set in.

The temperature may be lowered by drugs or by external applications. The drugs used are antipyrin, phenacetin, quinine, salicylate of soda, etc. The action of these drugs varies much in different individuals, but they usually lower the temperature. On the other hand, they tend to weaken the heart, so they must be used with caution.

The methods of lowering the temperature by external applications are the ice-pack, the ice-cap, the cold bath, and sponging with tepid water. Sponging with cold water is too painful. The ice-pack is probably the best method, as it is very effectual in lowering the temperature, and does not necessitate moving the patient to any extent. Cold acts as a stimulant in most cases, but if the heart should become weak, brandy and hypodermic injections of strychnia must be given. Pneumonia is not a bar to the employment of cold, but in old people,

and in persons with fatty hearts, it is contra-indicated.

The Preventive Treatment of Infectious Fevers.—
Prophylaxis.—The patient must be isolated as much as possible. A large well-ventilated room at the top of the house should be selected for the sick-room, and only the nurse and one relative should be admitted to it. All superfluous furniture, including carpets and curtains, must be removed, and a sheet must be hung before the door and kept constantly wet with 1 in 20 carbolic lotion. Everything which may be infected by the patient, such as spoons, cups, handkerchiefs, etc., must be placed in 1 in 20 carbolic before it leaves the room.

Everything intended for the sick-room, or taken from it, should be left on a table outside the door, so that the servants do not come in contact with the nurse. After the patient has recovered, the linen and bedding should be sent away to be disinfected by heat. The room may be disinfected by burning sulphur in it, 2 lb. being sufficient for an ordinary room. The doors and windows must be closed, the keyholes, crevices, etc., being blocked up. The chimney must be also blocked, and the sulphur placed in an iron pot. The pot is then placed in a bath containing a little water, and the sulphur set on fire and allowed to burn itself out. The room should not be disturbed for six hours.

It is now necessary to notify all cases of infectious disease to the medical officer of health for the district. In the case of epidemics, especially epidemics of cholera, typhus, or small-pox, a sharp lookout has to be kept for cases in an early stage of the disease, and even house-to-house inspection among

the poor may be necessary in order to secure the early isolation of infected persons.

† TYPHUS FEVER.

Causes.—The immediate cause is probably a bacterium. The predisposing causes are over-crowding, deficient food, physical exhaustion, and mental depression. Consequently the disease used to be common in besieged cities, gaols, etc. It is now rare in England, on account of the improved sanitation, and it only occurs in epidemics. It is very infectious, the breath and exhalations of the patient and his clothing, etc., carrying the poison.

The incubation period is about ten days.

Symptoms.—These may be described under five headings: (1) The onset; (2) the rash; (3) the typhoid state; (4) miscellaneous symptoms; (5) the temperature chart.

1. The onset is acute. There is a severe rigor, vomiting, intense headache with giddiness, and pains all over the body. The temperature rises rapidly to 102° or higher, and all the febrile symptoms, *e.g.*, thirst, anorexia, etc., are quickly established.

2. The early symptoms persist, and the rash appears on about the fifth day on the epigastrium and back of the wrists, but sometimes all over the body. It consists of reddish spots (*maculæ*) or patches, which are not appreciably raised, and which at first fade on pressure. In a day or two hæmorrhage occurs into the spots, which become purple in colour, and do not fade on pressure. This is the true *mulberry rash*. The spots fade slowly, and, being hæmorrhagic, may be seen after death.

In addition to the above, a violet discolouration, called the *subcutaneous mottling*, may occur on the back and on the back of the limbs. It is like post-mortem staining.

3. From the beginning of the disease headache and giddiness are prominent symptoms. Mental confusion soon develops, and later on delirium and coma set in. The tongue becomes dry, brown, and tremulous; there is subsultus tendinum, etc.; in fact, in about a week the patient is deeply in the typhoid state (see p. 23), and bedsores are apt to form.

4. In typhus the face has a suffused, semi-cyanosed appearance, and the pupils are extremely contracted. The spleen may be enlarged, and a peculiar smell is said to be exhaled from the patient.

5. The temperature rises on the first day to about 103° , and keeps at that point without any noticeable remission, till about the seventh day, when there is usually a definite remission. After this the temperature rises again, sometimes less high than before, sometimes higher, and it thus keeps high until about the seventeenth day; when the crisis occurs the temperature becomes normal in a few hours, often with profuse sweating, and the patient feels quite well. On the other hand, death may occur at any time before the crisis, most often at about the tenth day. The pulse is proportional to the temperature all through the disease, and becomes weak and dicrotic in the later stages.

Complications.—These are merely those which are common to all severe fevers, *i.e.*, bronchitis, pneumonia, nephritis with albuminuria, etc.; but gangrene, parotid bubo (suppurative parotitis), etc., may occur.

Post-mortem Appearances.—Unless the rash be present, there is nothing characteristic, the organs merely presenting the appearances which are found after death from any bacterial disease—that is, the endothelial surfaces (endocardium, pleura, etc.) are blood-stained, and the organs (lungs, spleen, etc.) are congested and softened. Complicating lesions, such as pneumonia, would, of course, be found if they existed.

For the **Differential Diagnosis**, see Typhoid Fever.

Treatment.—This must be expectant. Good nursing, and antipyretic and stimulant treatment, must be relied on. To prevent the spread of the disease, the isolation of the patient must be as absolute as possible, because proximity to the patient constitutes the chief risk of infection. The clothing, etc., must be burnt or disinfected.

† TYPHOID FEVER OR ENTERIC FEVER.

Cause.—The disease is due to a bacillus, which can be found in Peyer's patches before ulceration, in the mesenteric glands, and in the spleen. It is a short, thick bacillus with rounded ends, and it is motile. The disease is only communicable from one person to another by swallowing the bacilli which exist in the stools of the infected person.

The poison leaves the infected body in the stools, and it is said that when first passed the stools are not infectious, but become so later. The stools may soak from privies or from the ground into wells or streams, the water of which is in use as drinking water, or they may leak from broken drains into wells, or, by defective trapping, sewer gas may rise and be absorbed by drinking water in

cisterns, or by cans of milk in dairies. Milk may also be infected by the admixture of infected water, or by washing the cans with such water. Then when the infected water or milk is swallowed by a new individual the symptoms develope. Other articles of diet, *e.g.*, watercress, oysters, etc., have been supposed to be infected by sewage, and to carry the disease.

Typhoid fever is most common in young persons, and in the autumn, when the rains come after a dry season and wash the various impurities, which had become dried, into the nearest wells and streams.

The incubation period is about twelve days.

Symptoms.—The onset is gradual. There are slight chills, with headache and pains in the limbs for about a week. The temperature rises 2° every night, and falls 1° every morning, so that in a few days it gets to be 102° or 103° . The thirst, anorexia, quick pulse, and other febrile symptoms, develope slowly. Epistaxis may occur at this stage. Constipation may be present, but sometimes even at first there is a tendency to diarrhoea and abdominal pain. Early bronchial catarrh is not uncommon.

During the second week the patient usually takes to his bed. The headache may cease, but the abdominal symptoms are now definite. The *diarrhoea* consists in four or five large motions a day. They are passed without much pain, and are *yellow and pulpy*, like split-pea soup. At the same time, there is *tenderness*, and sometimes *pain*, in the right iliac fossa, and there may be gurgling on pressure there. The abdomen is full without being distended. In a few cases the constipation persists.

On about the tenth day the *rash* appears on the abdomen and chest. It consists of pink, oval, or round, slightly raised, spots, which always fade on pressure. The spots may be few or many, but they come out in crops; *i.e.*, a certain number appear simultaneously, last three or four days, and then disappear altogether. Several successive or inter-current crops may come out. The rash is less marked in children.

All this time the temperature keeps at about 102° to 104° , but there is usually a slight remission each day. The pulse is proportional to the temperature, and gradually becomes weak and dicrotic.

At the end of the second week the patient begins to get into the *typhoid state*, the tongue becoming dry and brown, and the mind confused. There is often deafness, and the spleen becomes large enough to be felt, and is tender.

In the early part of the third week the patient may be deeply in the typhoid state, with a tremulous tongue, delirium, coma, carphology, etc. The evacuations may be passed into the bed, and bedsores may form, and it is at this time that the grave complications, such as perforation, hæmorrhage, pneumonia, and nephritis, are most likely to supervene, and that death most commonly occurs.

In favourable cases, at the end of the third week *lysis* begins. The remissions in the temperature become more marked, and the rises less high. After a few days the remissions touch the normal, so that the fever is really intermittent, and gradually the remissions become subnormal, and the rises only reach the normal, so that defervescence is complete on about the twenty-fifth day.

Typhoid fever sometimes *relapses*, *i.e.*, after some days of apyrexia the disease comes on again. The relapse may be slight and short in duration, but sometimes the disease runs its whole course over again, with all the characteristic symptoms. There may even be a second relapse.

The return of strength after typhoid is usually slow, and patients may become infected with tubercular bacilli, and develop phthisis during convalescence.

A rise of temperature during convalescence may be due to: (*a*) a true relapse; (*b*) constipation; (*c*) too early a return to solid food; (*d*) a late complication, such as phlebitis, periostitis, etc. Perforation also may occur during convalescence.

In a few cases typhoid fever is so mild that the patient does not take to his bed. This is called *ambulatory* typhoid. Perforation has been known to occur in these cases.

Complications.—Hæmorrhage from the ulcers; perforation of the intestine; peritonitis with or without perforation; bronchitis; pneumonia; pleurisy; nephritis; meningitis; phlebitis and thrombosis of the femoral vein; abscess in the mesenteric glands; periosteal abscess, which is most often on the shin; parotid bubo.

Symptoms of Hæmorrhage.—There is a sudden fall in the temperature without improvement in the other symptoms. The pulse becomes quicker than before, and very small. The skin is cold and pale, and the respirations quick and sighing. Lastly, the blood may be seen in the stools. In slight cases the last may be the only sign of hæmorrhage.

Symptoms of Perforation.—There is a sudden

pain in the abdomen, with collapse, during which the temperature may fall; but very soon the patient shivers and vomits, the temperature runs up very high, and the whole abdomen becomes painful, tender, and distended from peritonitis.

The immediate *causes of death* in typhoid are: (1) simple asthenia, *i.e.*, gradual exhaustion, death usually occurring in the typhoid state; (2) perforation and peritonitis; (3) hæmorrhage; (4) pneumonia or other ordinary complications.

Pathological Anatomy.—The lesion consists in an infiltration of Peyer's patches, and often of the solitary glands as well, with leucocytes. This makes the patches swollen. The leucocytes and the lymphoid corpuscles in the patches die, and the mucous membrane over the patch also dies and sloughs away. Then the dead cells escape from the patch, leaving an ulcer. If recovery takes place, the mucous membrane grows over the ulcer, and no fibrous contraction of the scar occurs, so stricture of the bowel never results. Perforation may occur as follows: (*a*) By an extension of the ulceration through the muscular and peritoneal coats at one point, causing a pinhole opening; (*b*) by infiltration of the whole thickness of the gut beneath the patch with leucocytes, and subsequent sloughing, causing a large opening; (*c*) by accidental tearing of the floor of the ulcer by a piece of food, or by a sudden movement of the patient. In the first case, peritonitis may occur before perforation, and fibrinous adhesion of a neighbouring piece of bowel to the floor of the ulcer may prevent extravasation of fæces, and save the patient's life. In the other two varieties death is almost certain to occur.

At the **Post-mortem**, if death, as is common, occur in the third week, the ulceration of Peyer's patches will be found in the ileum. The ulcers are oval, with the long axis in that of the bowel; they are situated furthest away from the mesenteric attachment, and their base is smooth. In the most recent ulcers the edges are undermined, and sloughs may still remain attached to the floor. Higher up the patches may be swollen, but not yet ulcerated. The mesenteric glands are large, soft, and dark red, but they may have yellow points of suppuration in them. The spleen is dark, large, and soft. All the organs are usually congested and soft, and pneumonia and other complicating lesions may be found. In the rectus abdominis, adductors, etc., Zenker's degeneration (coagulation necrosis) of muscles may be found, but it is not peculiar to typhoid.

Treatment.—Typhoid is sometimes treated *radically* by mercury (in the early stages before ulceration), or by chlorine, salol, and other antiseptics, in the hope of killing the bacillus; but the expectant treatment is more commonly adopted.

The food must consist of milk and beef-tea. If there be much diarrhœa, milk only must be given. Brandy must be given if the pulse gets weak, as much as 12 oz. a day being well borne by the patient. Strychnia and digitalis are also good for weak heart action.

Hæmorrhage should be treated by ordering ice to be sucked, or placing an ice-bag, or if that fails a mustard plaster, on the abdomen. Internally, ergot, tannic acid, acetate of lead, and turpentine, are recommended, and opium may be necessary. Enemata of raw beef juice are useful in restoring the patient's strength.

Perforation can only be treated by opium, but abdominal section has been tried with the object of closing the opening.

Excessive diarrhœa may be treated by bismuth, chalk, hæmatoxylon, etc., with or without opium. Starch and opium enemata are useful.

Lung complications may be treated by ammonia, ether, etc., and by turpentine stupes over the chest.

The *antipyretic treatment* is highly commended in typhoid. It should be commenced early, before the patient is in the typhoid state, but even after that it may be of great value. If this treatment be adopted, the temperature should be taken every six hours, and if it be found that it has *remained* over 103° for more than twelve hours, an ice-pack or cold bath should be ordered. In addition to this rule, a very high temperature, say of 106° , should be treated by cold at once. The antipyretic drugs may also be tried, instead of the ice-packs, if the latter are required frequently. The patient must be kept on a fluid diet for fourteen days after complete defervescence.

To prevent the spread of the disease, the stools should be at once immersed in 1 in 20 carbolic, and any soiled linen disinfected. The drains should be frequently flushed.

The Differential Diagnosis of Typhoid.

1. **From Typhus.** — The *resemblances* between typhus and typhoid are that they are both prolonged fevers, and in both the typhoid state may be present, and the symptoms of the typhoid state are so conspicuous that minor distinctions become obscured.

The *differences* are: (1) that typhus begins acutely with a very high temperature in a few hours, and typhoid gradually, the temperature not being very high until several days have elapsed; (2) that in typhus head symptoms are early and prominent, while in typhoid they develop about the end of the second week; (3) that in typhus constipation, and in typhoid diarrhœa, is the rule; (4) that in typhus the rash occurs on the fifth day, and soon becomes hæmorrhagic, while in typhoid it occurs on the tenth day, and consists of much smaller spots, which never become hæmorrhagic, and therefore always fade on pressure. After death the typhoid ulcerations are, of course, quite distinctive.

2. **From Acute Tuberculosis.**—In this disease (for particulars of which see p. 119) miliary tubercles develop rapidly and simultaneously in all the organs of the body. It may happen that there are not enough tubercles in any one organ to create diagnostic symptoms in that organ, so that the patient suffers most from general poisoning, and in this case the disease may simulate typhoid.

The *resemblances* are that in both there is a prolonged fever, with daily remissions, and a tendency to get into the typhoid state. In both there may be diarrhœa or the physical signs of bronchial catarrh.

The *differences* are: (1) that in tuberculosis the temperature chart as a whole is very irregular, high temperatures being possibly recorded for a few days, and low ones for the next few days (remissions, however, occurring in both cases), while in typhoid the temperature chart records a regular course; (2) that in tuberculosis tubercles may

be seen with the ophthalmoscope in the choroid; (3) that in typhoid there is a characteristic rash; (4) that in tuberculosis the temperature-pulse ratio is disturbed, high pulse-rates accompanying moderate temperatures, or *vice-versâ*, while in typhoid it is fairly preserved; (5) that in tuberculosis the spleen is not often large enough to be felt, while in typhoid it usually is. Lastly, the presence of an old tubercular lesion in any part of the body, such as the vertebræ or other bones, or the glands of the neck, would point to tuberculosis.

3. From Tubercular Meningitis.—It occasionally happens, especially in children, that cerebral symptoms, such as squinting, contracted pupils, vomiting, etc., occur at the beginning of typhoid fever, and cause that disease to simulate tubercular meningitis. The spots, the enlarged spleen, the flaccid abdomen, etc., however, will show that the disease is typhoid, while the development of optic neuritis, retraction of the neck, rigidity of the abdomen, etc., would indicate that it is meningitis.

4. From Septicæmia.—If the site of septic infection be obvious, such as a wound or the uterus after delivery, difficulties are not likely to occur; but if the site be obscure, as, for instance, in the septicæmia which follows ear disease or in ulcerative endocarditis, septicæmia may be mistaken for typhoid fever.

The *resemblances* are that in both there is prolonged fever, with daily remission; that in both the typhoid state may develop; and that in both the spleen may be large.

The *differences* are: (1) that in septicæmia the remissions of temperature are greater, and are

accompanied by sweating; (2) that there are no spots, and the diarrhœa is not typical; (3) that careful search may reveal the site of infection.

5. **From Appendicitis.**—For a day or two this disease may simulate typhoid, because in both there is fever, with pain and tenderness in the right iliac fossa.

The *differences* are that in appendicitis the temperature rises quickly; there is no headache, and very soon a tumour can be felt in the right iliac fossa. In typhoid, on the other hand, headache is an early symptom, the temperature rises very slowly, and the right iliac fossa is soft and yielding. The spots and other symptoms settle the diagnosis.

6. **From Trichinosis.** — At the time when the embryos of the *Trichina spiralis* are swarming in the intestines and through the intestinal walls, there is fever, abdominal pain, and tenderness and diarrhœa, so that the affection may simulate typhoid. But the onset is more sudden than that of typhoid, the temperature chart is not so regular, and the eventual development of pain and swelling in the muscles is a conclusive sign that trichinosis is present.

FEBRICULA.

This is the name given to an attack of fever which lasts two or three days without developing any of the symptoms of the characteristic fevers. Sometimes the attack suggests that the fever may be the beginning of typhoid, but its abrupt termination in a few days dispels the idea. Other cases may be influenzal.

RELAPSING FEVER—FAMINE FEVER.

Cause.—The *exciting* cause is a spirillum, *i.e.*, a bacterium which is twisted so as to form a short spiral. It is found in the blood during the height of the disease, but disappears just before the crisis occurs. The *predisposing* causes are deficient food and bad sanitary surroundings. The disease is contagious.

Symptoms.—The disease begins with a rigor and a sudden rise of temperature to 103° or higher. Vomiting is common, and the pulse may be extremely rapid, but soon becomes weak. There are headache, febrile urine, thirst, and all the febrile symptoms. Jaundice often sets in on the fifth day, and in bad cases hæmorrhages from the stomach, nose, etc., or into the skin (petechiæ), etc., may occur. Constipation is the rule. Delirium and the symptoms of the typhoid state are rare.

The fever lasts about a week, and ends abruptly by *crisis*, with profuse sweating. During the next week there is no fever, but after that the temperature rises again suddenly, and all the symptoms recur. This relapse lasts four or five days, and then ends by *crisis*. After a few more days of apyrexia a third relapse may occur, but this usually lasts a shorter time, and after its conclusion by *crisis* convalescence is usually uninterrupted.

The disease produces great exhaustion, but if the patient is properly fed it usually ends in recovery. A peculiar form of conjunctivitis, with loss of vision, is a common sequela to the disease, and this, too, usually passes off if good food can be administered.

There are no characteristic post-mortem appear-

ances, but the organs, particularly the spleen, are found to be congested and large and soft.

The treatment is expectant.

THE EXANTHEMATA.

This is the name given to a class of fevers in which an eruption, or breaking out, is a prominent symptom. The class comprises scarlet fever, measles, German measles, chicken-pox, and small-pox.



Scarlet Fever.

Causes.—The exciting cause is a specific poison, the exact nature of which is unknown, although it is probably a bacterium. The disease is most common in young persons. Scarlatina is merely another name for scarlet fever, and does not imply a milder form of the disease. The disease is highly infectious.

The incubation period is about five days.

Symptoms.—The onset is acute. The disease begins with sore throat and general malaise, but in a few hours rigors, with severe headache and vomiting, occur, and the temperature rises to 103° or higher. At this stage the tonsils and fauces are swollen and red, and the cervical glands are large and painful. The pulse is quick and full; there is thirst and complete anorexia, and the urine is febrile. The tongue is white, with enlarged red papillæ (the strawberry tongue).

The rash appears on the second day, first on the chest and neck, from which parts it gradually spreads, until the whole body is covered. It consists of multitudes of bright red points, which are very closely set together, and as the skin between

the points is also injected, the rash appears to be a uniform scarlet colouration of the skin. It is not appreciably raised, and it always fades on pressure. The reddened skin has a soft, moist appearance.

On the second day the throat is still very sore, and yellow points of muco-pus may be seen on the tonsils.

In ordinary cases, improvement begins on the fourth or fifth day. The temperature falls slowly, and should be normal by the end of the week, at which time the rash should have disappeared. The throat, too, should now be normal.

Desquamation begins in about a fortnight, and may last two or three weeks or more. The skin may peel in fine scales, or in large pieces, especially on the hands, where the skin is thick.

But in some cases the throat is severely affected, and prevents the disease running a favourable course. Lacunar and follicular abscesses form on the tonsils and burst, leaving ulcers which may spread deeply. Then the lymphatic glands in the neck become very large, painful, and tender from acute lymphadenitis. They may also suppurate, and, if not opened, will burst and leave unsightly scars. Again, the inflammation may spread from the throat through the Eustachian tube to the ear, setting up otitis media and perhaps mastoid abscess. Lastly, the throat may infect the lungs or the whole body with septic micrococci, so that the patient gets septic pneumonia or general septicæmia, and dies in the typhoid state on the fourth or fifth day. In some cases the swelling of the throat may render tracheotomy necessary.

Complications — Sloughing of the throat ; abscess of the cervical glands ; otitis media, leading perhaps

to necrosis of the temporal bone, thrombosis of the lateral sinus, meningitis and cerebral abscess; pneumonia from insufflation of septic matter or from septicæmia. Acute nephritis. Pericarditis, endocarditis, and inflammation of other serous membranes. Arthritis, which is called scarlatinal rheumatism, because the joints become swollen and painful.

The Causes of Death in Scarlet Fever.—(1) Death has been known to occur without any obvious cause, and at an early period in the disease. This event has been attributed to the intensity of the poison. (2) Death may occur in the first few days from asthenia, due to the throat lesion or the septicæmia produced by it. (3) In the second or third week death may occur from acute nephritis. The first indication of the nephritis may be general dropsy or suppression of urine and uræmia. (4) Death may occur several years after the scarlet fever from cerebral abscess, etc., directly traceable to the otitis media, which was caused by the fever and had never been cured.

Varieties.—(1) Simple; (2) Anginal, in which the throat symptoms are severe; (3) Malignant, in which there is great depression, and in which the rash may be hæmorrhagic; (4) *Scarlatina sine eruptione*, in which no rash occurs.

For the **Differential Diagnosis** see p. 58.

Treatment.—A poultice or compress must be put round the throat, and gargles of Condyl's fluid or chlorate of potash, etc., must be ordered. In children the throat may be brushed or sprayed with the same astringent lotions. If ulceration occur, glycerine and sulphurous acid (in equal parts) should be applied to the fauces.

Internally a mixture containing chlorate of potash (gr. x.), sulphate of quinine (gr. v.), dilute hydrochloric acid (℥ v.), in syrup and water, may be given. Wine or brandy may be required. A careful look-out must be kept for ear disease and acute nephritis, so that they may be treated as soon as they occur. During desquamation exposure to cold must be carefully avoided, as it may bring on the nephritis. The peeling may be helped by frequent hot baths containing Condyl's fluid. The soles of the feet often continue to peel long after the rest of the skin is normal; and it must be remembered that a case is supposed to be infectious as long as desquamation is going on.

+ Measles—Morbilli—Rubeola.

Cause.—Measles is a very infectious disease, due to some unknown specific poison. It most frequently attacks children.

The incubation period is from ten to fourteen days.

Symptoms.—The onset is moderately acute. The child feels ill, and appears to be getting a cold. Sneezing is frequent, and in the course of two or three days a *coryza* is fully developed, *i.e.*, there is redness of the conjunctivæ, and a profuse discharge of thin mucus from the eyes and nose. There may also be a cough due to bronchial catarrh. At the beginning there may be chilliness, followed by a temperature of 101° , and sometimes a convulsion may occur. The temperature usually goes on rising, so that on the third evening it may be 103° or 104° .

The rash appears on the fourth day, first on the forehead, at the roots of the hair, and behind the

ears. During the next two or three days it spreads until it covers the trunk, and finally the limbs. It consists of dusky red patches, which often have a crescentic shape, and are distinctly raised, and fade on pressure. The patches may be close together, or may have bits of white skin between them.

In favourable cases the temperature begins to fall about the fifth or sixth day, and may reach the normal rapidly. The rash begins to fade about the same time, and should have disappeared by the eighth or ninth day. Its disappearance may be accompanied by much itching, and may be followed by fine branny desquamation. The favourable course of the disease may be interfered with by lung complications, or in rare cases by hyperpyrexia or a hæmorrhagic rash.

Complications.—Capillary bronchitis and bronchopneumonia, with lobular collapse and compensatory emphysema; laryngitis; pleurisy or emphysema; purulent conjunctivitis, going on to sloughing of the cornea; otitis media, which sometimes leads to necrosis of the temporal bone. In debilitated children cancrum oris may occur.

The Causes of Death in Measles.—(1) Death from the severity of the poison, causing sudden heart failure, or hyperpyrexia, or a hæmorrhagic rash; (2) bronchitis and its consequences; (3) cancrum oris.

Varieties.—(1) Simple; (2) Malignant (hæmorrhagic, etc.); (3) Morbilli sine catarrho, in which there is no coryza; (4) Morbilli sine eruptione, in which there is no rash.

For the **Differential Diagnosis**, see p. 58.

Treatment.—The patient must be kept in bed, and a diaphoretic mixture given as a febrifuge. A

good formula consists of liq. ammon. acetat., \bar{z} ii.; vin. ipecac., ℞x.; spir. æth. nitros., \bar{z} i.; aq. camph. ad \bar{z} i. This may be given every six hours, the dose being modified in the case of children. Sponging with warm water often relieves the discomfort of the skin. The lung symptoms must be treated as such, and in malignant cases wine and brandy must be given.

During convalescence, exposure to cold must be carefully guarded against, and cod-liver-oil and tonics may be given.

+ Rötheln—German Measles.

Causes.—This is a specific infectious disease due to an unknown poison. It is not a modified form of either measles or scarlatina, and an attack of it does not protect the patient from subsequently getting measles or scarlet fever. On the other hand, it must be admitted that the disease presents very varying characters, and that in small epidemics of it, in which all the cases are infected from a single source, hardly any two cases have the same symptoms.

The incubation period is from twelve to twenty days.

Symptoms.—The onset is rather acute, and may be severe, with shivering and vomiting and considerable fever. Sometimes there is nasal catarrh, but more frequently sore throat. In other cases, again, no special symptoms occur until the rash appears. The usual anorexia, thirst, quick pulse, etc., are present, and are proportional in their severity to the height of the temperature.

The rash may appear on the second, third, or

fourth day. It is often like a scarlatinal rash, with projections like those of urticaria on it, but it may be merely a red blush; or, on the other hand, there may be papules like those of measles. It may appear on any part of the body, and may be limited in extent or cover the whole body. It may itch intensely. The rash may last four or five days, and the amount of desquamation is very variable.

There are no special complications, and recovery nearly always results. The treatment consists in giving a diaphoretic mixture (see p. 49) and in keeping the patient in bed. An aperient at the outset may be advisable.

Varicella—Chicken-pox.

Cause.—This is a specific infectious disease due to some unknown poison. It is not a modified form of small-pox, because (1) an attack of it does not prevent a patient from subsequently getting small-pox; (2) it cannot be inoculated, while small-pox can readily be inoculated; (3) the papules of chicken-pox become vesicles in twelve hours, while those of small-pox become vesicles in three or four days.

The period of incubation is four to fifteen days.

Symptoms.—The onset is fairly sudden, but the symptoms are not severe. There is slight chilliness, with headache and a rise of temperature to about 101°. Within twenty-four hours a number of small papules break out in various parts of the body. In some cases the papules occur in successive crops on the trunk first, and then on the limbs and head. The face is not, as a rule, much affected. In another twenty-four hours, or earlier, each

papule becomes a vesicle, with clear fluid inside it. The vesicles are not umbilicated as a rule, and have no red areola round them. They very rarely become confluent.

In about three days after their appearance the vesicles dry up and form scabs. When the scabs come off, a red stain is left, which soon fades, and pitting very seldom occurs.

Treatment.—A saline aperient to start with, and a diaphoretic mixture afterwards, are usually all that is necessary. Children should be prevented from picking the scabs, as scarring may result if they do.

✕ Variola—Small-pox.

Cause.—This is a specific infectious disease due to some unknown poison. Although it is almost certainly due to a micro-organism, none of the numerous bacteria found in the pocks have been proved to be the cause of the disease. If a healthy person be inoculated with lymph from a small-pox vesicle by one of the methods used in vaccination, he contracts the disease, but in a milder form than if he had been infected in the usual way.

The incubation period is almost exactly twelve days.

Symptoms.—The onset is acute. There is a severe rigor, which may amount to shuddering. Vomiting may occur, the thirst is most distressing, and the headache is intense; but in particular there is great *pain in the back*, and to a less extent in the limbs. The temperature rises rapidly to 104° or higher, the pulse is rapid and full, the urine is febrile, and the bowels constipated. This primary fever, as it is called, lasts without remis-

sion until the true rash comes out, but during this period an *erythematous rash* may appear on the face or trunk, and in certain bad cases this early rash becomes hæmorrhagic.

The true variolous rash comes out on the third day, first of all on the face, especially on the forehead. Pocks may also be seen at an early period on the soft palate. On the following days successive crops of pocks occur on the chest and limbs, but the face is nearly always the part most affected.

The rash begins as an eruption of papules. Each papule is at first red, and it soon gets hard, and feels like a shot in the skin. It gradually becomes paler, and on about the fourth day it can be seen that a little clear lymph is accumulating in it. On the sixth day, *i.e.*, on the ninth day of the whole illness, the papule has become a perfect vesicle, which has two chief characteristics: one is that it is umbilicated, *i.e.*, it has a depression in the middle of its summit; and the other is that it is multilocular, *i.e.*, there are little septa, which divide the vesicle into separate spaces, each of which is filled with lymph. On the seventh day of the rash the clear fluid becomes turbid, and shortly afterwards it is converted into pus, on which the umbilication and loculation disappear, and the vesicle becomes a pustule, which is surrounded by a red areola. The pustules either burst or dry up to a scab. The scabs separate in about a fortnight, and leave a reddish stain, which lasts for a long time. Eventually a depressed white scar or pit is left in the position of the pock.

On the day on which the rash appears, *i.e.*, the third day of the disease, the temperature falls to

the normal, and the patient feels quite well. This is unfortunate, because he is apt to think nothing of the spots on his forehead, and to leave his home and infect other persons. During the maturation of the pocks, the temperature keeps normal, until suppuration occurs on the tenth day. Then the temperature rises again, and the *secondary* fever sets in. This secondary fever is proportional to the severity of the attack, and, in fact, to the number of pocks. In a mild case the fever is not very high, and does not last very long. In a bad case the temperature keeps at 103° or higher; complications, such as pneumonia and nephritis, are apt to occur, and the patient gets into the typhoid state, with a dry, brown, tremulous tongue, delirium, coma, etc. He is very likely to die, but may recover.

Complications.—Pneumonia; bronchitis; pleurisy; nephritis, causing albuminuria; conjunctivitis, and perhaps destruction of the eye from the formation of a pock on it; erysipelas between the pocks; gangrene. Practically nearly any complication may occur.

Varieties.—(1) Discrete, in which the pocks do not touch; (2) Coherent, in which they touch but do not coalesce; (3) Confluent, in which the vesicles and pustules unite to form large blebs; (4) Hæmorrhagic or malignant, of which there are two sub-varieties: (*a*) hæmorrhages may occur into the pocks; (*b*) the early erythema is replaced by extensive hæmorrhages into the skin or from the mucous membranes, and there may be so much poisoning that the patient may die of asthenia before the true rash appears.

The **Prognosis** is of course worse in old or very

young persons, and in the confluent and hæmorrhagic varieties of the disease.

The Histology of the Pock.—The first event is necrosis and cloudy swelling of a group of epithelial cells in the rete Malpighi of the skin. This mass of dead cells, with a few leucocytes and dilated capillaries, constitute the papule, and the hardness of the dead cells makes it shotty. Then the lymph is exuded into the papule, and as the fluid forces its way between the closely-adherent epithelial cells, the latter cling together here and there, and form septa between the collections of fluid, whence the loculation. The umbilication is due to the presence of a sweat-gland or hair in the centre of the pock. On the tenth day of the disease (seventh of the rash) there is a fresh exudation of leucocytes, which converts the lymph into pus. The leucocytes also infiltrate, and destroy the adjacent cutis vera, whence the *pitting*.

For the **Differential Diagnosis**, see p. 58.

The Treatment must be expectant. In the early stages an aperient may be given, followed by the diaphoretic mixture. The pains in the back may be treated by warm applications; and lemonade, milk-and-soda, etc., may be given to relieve the thirst. In the secondary fever stimulants must be given freely, and antipyretics may be tried.

As regards the pocks, it would be of great advantage if each pock and its contained micro-organisms and toxins could be destroyed before they re-infect the blood. With this object, it has been recommended to inject pure carbolic into them, or to open each one and touch the base with solid nitrate of silver. But if the pocks are many this becomes an impossible task. Consequently we have to be

content with keeping the pocks and scabs as aseptic as possible by frequent bathing with antiseptic solutions.

Modified Small-pox.

As has been stated, small-pox produced by inoculation runs a milder course than if it be acquired by inhaling or otherwise absorbing the poison from an infected person. A papule forms at the site of inoculation, and this becomes a vesicle, and finally a pustule on the seventh day after inoculation. On this day there is a rigor, followed by a rise of temperature, by vomiting, and by pain in the back. Then on the tenth day after inoculation, *i.e.*, the third day after the rigor, the true papules appear on the face, just as in ordinary small-pox. The eruption runs its course, and the secondary fever takes place as usual, but the whole disease is, as a rule, of a mild type.

When small-pox occurs in a person who has been vaccinated, the symptoms are also nearly always modified, and this modified form is called *varioloid*. The modifications are of various kinds. For instance, the primary fever may be mild, and the pocks few. Also the pocks may abort, *i.e.*, they may become absorbed either in the papular or vesicular stage, and not become true pustules; or sometimes vesiculation and pustulation occur very rapidly, so that scabs form in a few days. As a consequence of the mildness of the eruption, the secondary fever is slight.

Vaccinia—Cow-pox.

This is a disease which affects cows and calves, and is usually met with in the form of vesicles on

the udder. It is believed that the disease is really small-pox, but that in cows small-pox is so modified as to be a very mild affection.

Vaccinia can be inoculated into human beings by means of the lymph taken from the vesicles, the process being called *vaccination*; and after inoculation it runs a definite course, which will be described below, and is practically always a trivial disease. Further, it has been conclusively shown that a human being who has had vaccinia is protected against small-pox just as if he had had that disease itself.

The protection against small-pox afforded by vaccination does not last for ever. Hence persons who have been vaccinated in infancy must be vaccinated again at puberty. This is usually sufficient, but if the person be likely to come in contact with small-pox, as a doctor or nurse, etc., he or she should be revaccinated a third or even a fourth time. If, as sometimes happens, a vaccinated person gets small-pox, the disease usually runs a mild course (see Varioloid, p. 55).

Vaccination.—A child should be vaccinated when it is six weeks old. It should be examined first to see if it be quite healthy, and if not, the operation may be postponed, unless small-pox be prevalent, in which case nothing but a serious ailment would justify the postponement. On the other hand, if the child, although healthy itself, be exposed to the infection of scarlet fever, diphtheria, or septicæmia, etc., the vaccination should be delayed for a time.

The **lymph** may be drawn up in capillary tubes from the vesicles of the calf or of the human being, or it may be placed on ivory points and allowed to dry. Lymph so collected may be kept for a long

time without losing its efficacy. In the *arm-to-arm* method of vaccination the lymph is taken direct from a vesicle on the point of a lancet, and immediately inoculated into the arm of the person to be vaccinated. Whether the lymph is to be kept or to be used at once, the essential point is that it should be quite clear and free from turbidity, *i.e.*, it must be collected before suppuration has commenced. In the vaccination vesicles of the human being, it will be usually found that the vesicle is fully formed and filled with clear lymph on the eighth day after vaccination, so that is the day which should be selected for collecting the lymph. By the ninth day turbidity will have commenced. In all cases it is essential to see that the child from which the lymph is taken is healthy, and in particular that it is not syphilitic.

Various methods of inoculation are used. A number of fine scratches or minute punctures may be made in the skin with a lancet, and the lymph rubbed into them. Three or four inoculations are necessary, and it is usual to make them close together on the skin over the upper part of the deltoid in one or both arms.

The lancet should be sterilized before use in the flame of a spirit-lamp, and the skin should be carefully washed. If dried lymph be used, it must be moistened with distilled water or glycerin.

Development of the Pock.—On the second or third day red papules appear at the site of inoculation, and these soon show signs of becoming vesicular. On the fifth day the vesicles are distinct to the naked eye, and on the eighth morning they are perfect, and still contain clear lymph. As a rule, the centre of the pock is depressed and the circum-

ference raised and filled with fluid. On the eighth evening or ninth day a red areola of inflammation encircles the pock, and the contents of the vesicles become turbid. The patient may have a headache and slight pain. On about the eleventh day the vesicles burst and dry up, forming scabs which fall off about the twentieth day. The scar which is left soon becomes white, and it is depressed, and has minute pits on its surface. This is the characteristic *vaccination mark*, and its presence is a rough test in after-life of the efficiency of the vaccination.

The Differential Diagnosis of the Exanthemata.

Scarlet Fever.—(1) From *acute tonsillitis*. In both, enlarged secreting tonsils and sudden fever occur, but vomiting is rare in tonsillitis and common in scarlet fever, and in twenty-four hours the appearance of the rash settles the diagnosis.

(2) From *measles*. The raised crescentic portions of the measles rash are quite different from the diffuse smooth punctiform rash of scarlet fever.

(3) From *German measles*. The sore throat is not so severe as that of scarlet fever, and the fever not so high, and vomiting is rare. The rash of *rötheln*, too, comes out later, and usually has papules on it, which is not the case in scarlet fever.

(4) From *small-pox*. Difficulty can only occur in the early stage of small-pox, when an erythematous rash like that of scarlet fever may be present. In both, rigors, vomiting and high fever occur, but, as a rule, in scarlet fever the *sore throat* is the prominent symptom, and in small-pox the *pain in the back*. Once the true papules of variola appear on the third day, all difficulty ceases.

(5) From *ptomaine-poisoning*. In this affection

the rash is practically identical with that of scarlet fever, but it does not begin on the chest and spread all over the body in a regular manner, but may occur on any part and to a very variable extent. The sore throat and vomiting of scarlet fever are usually absent, and the amount of fever is very variable.

Ptomaines are animal alkaloids which may be formed by degeneration of the tissues, but are often produced by the decomposition of retained fæces. In either case they may be absorbed into the blood, and the rash described above is attributed to their presence there. In practice these rashes may occur after the use of enemata in chronic constipation, and it is supposed that the water, by softening the dried fæces, allows the intestine to absorb the ptomaines contained in them. This rash has also been known to occur in intestinal obstructions and appendicitis. As regards degeneration of the tissues, the rash which sometimes occurs in virulent cases of septicæmia resembles the ptomaine rash.

(6) From *belladonna-poisoning*. In belladonna-poisoning a rash like that of scarlet fever may occur, and the throat may be painful from being dry. But the rash is not so constant in its position and extension as that of scarlet fever, and the throat is not swollen and secreting. Also there is not the same high fever and vomiting. In belladonna-poisoning, also, the pupils become dilated, and there may be delirium.

Measles.—(1) From *scarlet fever*. See above.

(2) From *small-pox*. In both there is fever for two or three days before the rash appears, and in both the rash first appears as pink papules on the forehead. But the fever, rigors, etc., are much more severe in small-pox, the *pain in the back* is intense,

and if there be coryza, it is much less marked than that of measles. Then, the rash of small-pox comes out on the third day, and that of measles on the fourth, and the papules of small-pox become *shotty* in a few hours, which never occurs in measles.

(3) From *German measles*. In both there is fever, and the rash may be papular. But in R \ddot{o} theln the rash appears long before the fourth day, and does not usually begin on the forehead. Also the sore throat is more marked in R \ddot{o} theln than in measles.

(4) From *influenza*. In both there is coryza, but in influenza the onset is usually more acute, and accompanied by pains in the back and limbs. In measles the symptoms of onset become steadily worse until the rash comes out; and the appearance of the latter settles the diagnosis.

(5) From *copaiba-poisoning*. In both there is coryza and a rash composed of raised red patches. But in copaiba-poisoning the rash does not begin on the forehead and spread thence, as in measles, but may be quite irregularly distributed. Also there is no fever to speak of, and the breath and urine of the patient may smell of copaiba.

(6) From *arsenical poisoning*. In both there may be coryza and a red papular rash, and it may be difficult to settle the diagnosis unless a history of arsenical poisoning can be obtained. But in measles the fever and constitutional disturbance is greater, while in arsenical poisoning there may be vomiting or other signs of gastro-intestinal irritation.

Small-pox.—(1) From *scarlet fever* and (2) from *measles*. See above.

(3) From *chicken-pox*. The essential clinical difference is that in chicken-pox the papules become

vesicles in a few hours, while in small-pox vesiculation takes three or four days. But the three days' fever which precedes the rash, the pain in the back, and the general severity of the disease, usually make the diagnosis easy, except in the case of modified small-pox. But even in the latter case vesiculation does not occur so rapidly as in chicken-pox; there is always some primary fever, and the face is the favourite site for the pocks. Umbilication of the vesicles is uncommon in chicken-pox.

(4) From *pustular skin diseases*. In these the eruption does not run a regular course as in small-pox, and the characteristic primary fever is absent.

✦ ERYSIPELAS.

Cause.—The immediate cause is the *Streptococcus crysipclatis*, which is a micrococcus, the individual cocci of which are arranged in a row like a necklace of beads. It closely resembles the streptococcus of septicæmia. It enters the skin through some wound or scratch, but some think that it may infect an unbroken mucous membrane.

Symptoms.—The disease usually sets in abruptly with chills, or even rigors, and sometimes headache and vomiting. The temperature always rises to 102° or higher. In medical practice facial erysipelas is most often seen, so this will be described as a type.

The local symptoms usually begin at a mucocutaneous surface, *i.e.*, at the junction of the lips, nose, conjunctivæ, or ears, with the skin. There is first a red patch, which itches or burns. This increases until a considerable piece of skin becomes red, swollen, hot, and brawny to the touch. The direction in which the patch is spreading can always be recognised by the fact that the line of demarca-

tion between the red, swollen skin and the normal skin is abrupt, while where the inflammation is not extending the redness shades off gradually into the normal skin. The red patch is always painful and tender, and the neighbouring lymphatic glands become enlarged.

Facial erysipelas may affect both sides of the face, and usually spreads upwards. The eyelids become very much swollen and œdematous (because their subcutaneous connective tissue is loose), and the eyes may be completely closed. The scalp is always œdematous when affected. In other cases the erysipelas spreads downwards to the neck. The fever, with its quick pulse, etc., persists while the inflammation lasts, and declines with it. When it affects the limbs, the disease tends to spread upwards towards the trunk.

Erysipelas is sometimes recurrent, *i.e.*, after a few days it may appear to be subsiding, but suddenly there is a fresh extension of the inflammation and a fresh rise of temperature.

In many cases the epidermis is raised here and there by sero-purulent fluid, forming blebs or bullæ. In certain bad cases, called *phlegmonous erysipelas*, the subcutaneous connective tissue, and even deeper parts, become inflamed, and sometimes deep-seated abscesses are formed.

Complications. — Bronchitis ; pneumonia ; nephritis ; pharyngitis, and laryngitis by extension into the mouth ; meningitis, by extension through the skull by way of the veins of the diploë.

Prognosis.—This is favourable except in the case of the very old or the alcoholic. Death is usually due to some complication.

Pathological Anatomy.—The skin is infiltrated

with leucocytes and a little serum, and in bad cases with fibrin. The cocci are found in the lymphatic vessels, just beyond the line of demarcation of the inflammation, but not in the older parts. They are massed together so as to form solid cylinders which plug the lymphatics.

Differential Diagnosis.—Facial erysipelas might be taken for the swelling of the face, caused by abscess in the antrum. But in the latter case the bone is swollen, and not merely the skin, and examination inside the mouth may assist the diagnosis.

Treatment.—Begin with a powder containing 3 grains of calomel and 10 grains of pulvis jalapæ. Then prescribe tinct. ferri. perchlor. (M xx.), with quinine, dilute hydrochloric acid, and water, to be taken every sixth hour. Locally the inflamed part must be kept covered with lint soaked in lead lotion. For the face, a mask of lint may be made, and, to keep the lotion from evaporating, a second mask of oiled silk may be worn outside the lint. Other local applications which are recommended are: Painting the part with collodion and castor-oil, or with a solution of ergotine, or with perchloride of iron and glycerine; or dusting it with powdered starch and zinc oxide. Hypodermic injections of carbolic acid at the line of demarcation, or applying nitrate of silver there, have also been recommended.

ANTHRAX—MALIGNANT PUSTULE.

Cause.—This disease is due to the *Bacillus anthracis*. This is a large bacillus, about as long as the diameter of a red blood disc. It causes the splenic fever of cattle. Human beings become infected from cattle, especially men who flay cattle

or who handle the wool of infected animals. Hence the name *wool-sorter's disease*.

Symptoms.—There is an external and an internal variety of the disease.

The *external* form occurs after a scratch with an infected knife, or by the infection of a previously existing scratch. A slightly painful red pimple occurs in a few hours at the site of infection. This soon becomes vesicular, and in about three days it becomes a pustule, surrounded by red, indurated, swollen skin. This gave rise to the name *malignant pustule*. The pustule soon becomes a black eschar, and the red areola round it may become three or four inches in diameter, and secondary vesicles may form on it in a circle round the eschar. For a time during this process the patient may show very little constitutional disturbance, but at any moment the bacilli may enter the blood, and then rigors, high fever, and great prostration, with perhaps hæmorrhages from the mucous surfaces, will occur, and death will soon follow.

The *internal* form may affect the lungs or intestines chiefly. The pulmonary form is the common one in wool-sorters, who inhale the infected dust from the wool. There is inflammation of the bronchi and lungs, or of the intestines, in these cases, and ulcerated nodules may be found on the mucous membranes. Rigors, high fever, and rapidly fatal prostration occur, and hæmoptysis or melæna are common.

Diagnosis.—The pustule in the external form is characteristic, but the internal form could only be diagnosed by finding the bacilli in the blood or excretions, although the occupation of the patient would be suggestive.

Treatment.—The pustule should be excised freely as soon as its nature is known. The internal cases are practically hopeless.

GLANDERS—FARCY.

Cause.—This disease is due to a bacillus, called the *Bacillus mallei*. It is about as long as the tubercular bacillus, and a little thicker. Glanders and farcy are practically the same disease, the name of glanders being given when the disease affects the nose primarily, and that of farcy when there is general infection from the first, so that the lesions come out on the skin. The disease is one which affects horses, and human beings become infected by inhaling the breath of an infected horse, or perhaps by the discharge from the nose when the horse snorts.

Symptoms.—The disease may be acute or chronic.

Acute glanders begins with rigors, vomiting, and high fever, and at the same time there is a discharge from the nose (and sometimes from the eyes), which is at first thin, but soon becomes thick, dirty-looking, offensive, and often sanious. In a short time papules on the skin, and subcutaneous and submucous nodules, appear, which rapidly suppurate, leaving ulcers of considerable depth. The prostration is very great, and death in the typhoid state may occur in the course of a day or two. If the nose be not affected, the disease would be called acute farcy, and the rapidly-breaking down subcutaneous nodules, which are called farcy-buds, would be the chief lesion.

In chronic cases the symptoms are much the same, but the onset is more gradual, and the nasal discharge and nodular eruption are less. Acute

glanders is practically always fatal, but chronic cases may recover.

Microscopically, the nodules consist of masses of small round cells, into which hæmorrhage may occur. The skin around them may be erysipelatous.

The **Treatment** must be expectant. Quinine and stimulants may be given, and antiseptic dressings, particularly iodoform ointment, should be applied to the ulcers.

ACTINOMYCOSIS.

This is a disease which chiefly affects cattle. It is due to a fungus, called the *ray fungus* or *actinomyces*. This fungus is thought to be present on certain kinds of corn, and the cattle become infected by eating such corn. Consequently the lesions are most common in the mouth or on the jaws, but they may be found in the trachea, lungs, and alimentary canal.

The lesions consist of tumours composed of granulation tissue, and they may grow to a very large size. They frequently degenerate, and, until the discovery of the fungus, they were thought to be sarcomata. In the granulation tissue the fungus grows in groups, which form white specks, which are visible to the naked eye. Microscopically, the white specks are found to consist of filaments of the fungus arranged like rays of light, radiating more or less from a common centre. The most characteristic filaments are *clubbed* at one end.

In the human being the tumours formed by the fungus rapidly soften and break down. Hence they look like abscesses with crumbling walls, but the presence of the white specks in the pus is

characteristic. They may occur in the lungs, intestines, and liver.

The diagnosis depends on the discovery of the fungus, and the treatment consists in removing the growths if possible.

THE PLAGUE.

This disease is only met with nowadays in the East of Europe and in Asia. It is due to a short, thick bacillus. The predisposing causes are uncleanliness, overcrowding, and privation. It occurs in epidemics.

Symptoms.—The disease sets in with rigors, vomiting, fever, and great depression, and the patient may rapidly get into the typhoid state. Hæmorrhages from the mucous membranes or into the skin may occur. The inguinal lymphatic glands, and sometimes the axillary and cervical also, become large and painful, and soon suppurate, so that deep-seated abscesses result. This gave the name of *bubonic plague* to the disease. Carbuncles may also occur. The disease is very fatal, and no special treatment can be recommended.

DIPHTHERIA.

Cause.—The immediate cause is a bacillus, which is about the same length as the tubercular bacillus, but twice as thick. The disease is usually spread by inhalation of the breath of an infected person, but it is thought that the bacilli may be carried by sewer gas. The disease is most common in young persons, and it usually prevails in epidemics.

It has been proved that the formation of the membrane is due to the presence in the mucous

membrane of the bacillus, but that the general symptoms of the disease, such as the fever, albuminuria, and paralysis, are due, not to the presence of the bacillus in the blood, but to the toxins which are produced by the bacillus at the place where the membrane is formed, which toxins are freely absorbed into the blood, and carried all over the body.

The *diphtheria antitoxin* which is now in use is prepared from the blood serum of the horse. The bacilli are first made less virulent by cultivation in a special way, and then a small quantity of the cultivation is injected into the horse on several successive occasions. The effect is that the blood of the animal becomes charged with a substance which is an antidote to the diphtheritic toxin, and which is therefore called the antitoxin.

The incubation period is about four days.

Symptoms.—The disease begins with sore throat. There is a moderate amount of fever, and the pulse is quick and weak. Thirst, loss of appetite, and febrile urine, are of course present.

In about twenty-four hours a yellowish-white patch may be seen on either tonsil or on the soft palate. Usually there is only one patch, and it is very adherent to the mucous membrane. It looks like a piece of wash-leather, and if torn off forcibly it leaves a raw surface, on which the membrane soon forms again. In due course a patch may form on the other tonsil, and as the original patch enlarges, nearly all the fauces may become covered with membrane. The glands in the neck enlarge early, and they are painful and tender and hard. In a day or two albuminuria occurs. In a favourable case the membrane becomes detached in a few

days, and although a second membrane may form on the raw surface, it soon comes off, and no further formation of membrane occurs. The glands then subside, the albuminuria disappears, and the patient gets well. Even in mild cases, however, there is great exhaustion, and convalescence is very slow.

On the other hand, in many cases the membrane extends beyond the fauces, and the course of the disease is distinctly unfavourable.

1. The membrane may cover the pharynx and the posterior nares (post-nasal variety). There is then a dirty, offensive discharge from the nose, which excoriates the upper lip, and the patient has to breathe through the mouth. The glands in the neck get very large, and the whole neck may feel brawny. These cases last about ten days, so there is plenty of time for the absorption of toxins. Hence the amount of albuminuria becomes very great, the heart gets very weak, and the patient may die in the typhoid state or recover with great difficulty.

2. The membrane may spread down the larynx, trachea, etc. The first indications of this are hoarseness and a croupy cough. In children the membrane soon obstructs the larynx, and consequently stridulous breathing, sucking in of the lower ribs and episternal notch during inspiration, deficient entrance of air into the lungs, and cyanosis, become prominent symptoms, and call for the performance of tracheotomy. After tracheotomy the breathing is relieved, but as the membrane usually continues to spread down the trachea, the dyspnoea returns, and death is likely to occur from asphyxia. Sometimes fibrinous casts of the bronchi are expectorated. In adults a great deal of membrane

may be present in the larynx without causing dyspnœa.

3. Membrane is rarely found on the œsophagus, but it may form on the stomach, and cause vomiting. It occurs sometimes on the conjunctiva.

4. Membrane may form on any wound or raw surface on the patient's body.

The causes of death in diphtheria are : (1) sudden paralysis of the heart, which may occur at any time after the third or fourth day ; (2) asphyxia, from laryngeal obstruction before tracheotomy, or from bronchial obstruction or pneumonia afterwards ; (3) exhaustion, from toxæmia in the post-nasal cases ; (4) acute tubular nephritis ; (5) the late paralysis, which occasionally affects the heart.

The great sequela of diphtheria is paralysis. The cause of the paralysis is acute parenchymatous neuritis, which is due to poisoning of the nerves by the toxins. It usually begins with paralysis of the soft palate, which makes the voice nasal and allows the food to pass through the nose. On inspection, the palate is seen to be immobile, and it is anæsthetic. The eye is the part next affected, the muscles of accommodation being often paralyzed, causing loss of vision, and the oculomotor muscles, causing ptosis or strabismus. Afterwards any of the voluntary muscles may be taken, and sometimes the heart or the bladder. A very slow or intermittent pulse may indicate the approach of heart failure.

Pathological Anatomy.—The membrane is formed as follows : There is an exudation of plasma into the connective tissue beneath the epithelium of the tonsil or soft palate. This coagulates and deposits fibrin, with a mixture of leucocytes, in that posi-

tion. Then the epithelium above dies, and undergoes coagulation necrosis; *i.e.*, the epithelial cells lose their nuclei, become more or less hyaline, and are fused together to form a tough layer, which is closely adherent to the fibrin in the connective tissue beneath. So that the component parts of the membrane are dead epithelium above, and fibrin and leucocytes, resulting from exudation, below. The bacilli can be seen in it, and in addition septic organisms of all kinds may abound. In the trachea the membrane is distinct, and forms a complete cast of the tube. It is less adherent than on the fauces. In the bronchioles there is no distinct membrane, but the tubes are filled with a thick yellow fluid consisting of pus and threads of fibrin. In the lungs there is often a lobular pneumonia, the air-vesicles being filled with fibrin and leucocytes.

The glands show acute lymphadenitis, but are harder and paler than the mesenteric glands in typhoid, because the lymph follicles undergo coagulation necrosis and are converted into fibrin.¹

In the kidney the epithelial cells of the convoluted tubes lose their nuclei, and become granular and fatty. Some consider this a simple degeneration because the intertubular connective tissue is not infiltrated with leucocytes. Others think it a parenchymatous nephritis.

The nerves show swelling of the nuclei of the primitive sheath and breaking up of the myelin into drops. This is called a parenchymatous neuritis. There is no increase of neuroglia.

Treatment.—Although the antitoxin is not infallible, the evidence in its favour is so convincing that it must always be used, if it can be obtained. It produces rapid detachment of the membrane and

improvement in the toxic symptoms. Its use is sometimes followed, after an interval of about ten days, by an erythemathous rash and pains in the joints, but no harm seems to result from these symptoms. At the same time tinct. ferri perchlor. must be given in large doses. Quinine is also recommended, and some physicians give mercury internally.

Locally, common paraffin is the best application. It should be painted on to the membrane every three or four hours. Other good local applications are glycerine and sulphurous acid (āā), or glycerine of perchloride of iron, or sulphur in powder. Wine and brandy are always required, and tracheotomy must be performed as soon as it is clear that the larynx is affected. To prevent infection, everything that touches the patient's mouth or nose must be disinfected at once, and nurses should avoid inhaling the breath.

Differential Diagnosis.—The presence of a definite adherent membrane distinguishes diphtheria from other forms of sore throat. If the appearance of the throat be not quite characteristic, the presence of albuminuria may show that the disease is diphtheria. In some cases it is only possible to be sure of the diagnosis when the diphtheritic bacillus is found.

MUMPS—IDIOPATHIC PAROTITIS.

Cause.—This is a specific infectious disease due to an unknown poison. It is most common in young persons, especially males. It is therefore apt to occur as an epidemic in boys' schools.

Symptoms.—The disease begins with a pain on moving the jaw, which is soon found to be due to enlargement of the parotid gland on one side. The

gland continues to swell, and soon fills up the hollow behind the ramus of the lower jaw, while the socia parotidis produces a swelling over the masseter. Movements of the jaw and swallowing are now very painful, and there is always a certain amount of fever. In a day or two the other parotid swells in the same way, and as the submaxillary glands may also swell, the jaw and neck may become encased in a collar of enlarged glands, which gives the lower part of the face a swollen and grotesque appearance. The affected glands are always painful and tender, but they never suppurate. The lymphatic glands may become enlarged. The swelling begins to subside in a few days, and should be gone by the tenth day.

In some cases orchitis occurs during or after the parotitis, and it may cause atrophy of the testicle. In women ovaritis or inflammation of the breasts is said to occur occasionally. These inflammations are said to occur by *metastasis*. As recovery practically always occurs, it is difficult to say what the pathological anatomy may be. It may be an interstitial parotitis with an exudation of leucocytes and serum into the connective tissue between the acini, or a parenchymatous parotitis with swelling of the epithelial cells which line the acini.

Treatment.—Begin with a calomel purge, and then prescribe saline diaphoretics. Locally, various poultices, or stupes, may relieve the pain in the parotids.

Differential Diagnosis.—The swelling of the socia parotidis in front of the masseter will distinguish inumps from enlarged lymphatic glands. Any acid fluid, such as vinegar or lemon-juice, if placed in the mouth, causes a sharp pain in the parotids

during mumps, and this may be of diagnostic importance in doubtful cases.

PERTUSSIS—WHOOPIING-COUGH.

Cause.—This is a specific infectious disease due to an unknown poison. It is most common in children.

The period of incubation is from two to six days, or longer.

Symptoms.—There are two characteristics of this disease: (1) a catarrh of the respiratory passages; and (2) paroxysms of cough, complicated by spasms of the larynx. It is doubtful whether the poison causes an irritable condition of the mucous membrane of the larynx or an irritable condition of the vagus nerve. In either case a tendency to laryngeal spasm would be present.

The disease begins as an ordinary cold and cough, such as might mean a simple bronchial catarrh. Then after a variable time the cough assumes its characteristic paroxysmal character.

The Paroxysm.—The child suddenly gives a gasp, and then emits a rapid succession of coughs without any interval for inspiration, until it gets blue in the face. Then it *must* inspire, but as the larynx is still spasmodically contracted, it has to inspire through a narrow chink, so that a loud whoop results. Sometimes vomiting occurs. A number of paroxysms may follow each other consecutively. Sometimes there may be two or three paroxysms every hour, sometimes only one or two a day. In the intervals of the paroxysms the child breathes easily, and there is no aphonia, which shows that there is no disease of the larynx.

During the paroxysms various accidents may

occur (see Complications). In some cases the catarrh remains mild, but in others severe bronchitis and its consequences ensue.

The disease may last six weeks or three months or more, but recovery occurs, unless serious complications supervene.

Complications.—Bronchitis, broncho-pneumonia, lobular collapse, and compensatory emphysema. Permanent emphysema may result from severe whooping-cough in children. The accidents which may occur during the paroxysm are subconjunctival hæmorrhages; epistaxis; cerebral hæmorrhage (very rare); rupture of the membrana tympani; rupture of the air-vesicles, causing pneumo-thorax; umbilical or inguinal hernia; prolapsus ani.

An ulcer at the frænum linguæ is often found in whooping-cough, probably from laceration during the cough.

Treatment.—The object is (1) to prevent the lung complications, and (2) to diminish the number and frequency of the paroxysms. To attain the first object, exposure to cold should be carefully avoided, and if it be winter-time, the child should remain indoors. If marked bronchitis occur, it must be treated as such. For the second object, antispasmodic drugs, such as tinct. belladonna (℥ v.), bromides, ipecacuanha, and even chloral (gr. v.), must be given. Alum and simple alkalies are also recommended. The number of the paroxysms per diem should be counted, so that the effect of the drugs can be estimated. A good method of treatment is to keep the air of the sick-room saturated with a carbolic spray, and the odour proceeding from gasworks is said to be efficacious in stopping the paroxysms in chronic cases.

SEPTICÆMIA—PYÆMIA.

Cause.—This disease is caused by the *Streptococcus pyogenes*, or the *Staphylococcus pyogenes aureus* (see p. 4). There are differences in the action of these two varieties of cocci, but when the blood becomes infected with either of them the disease known as septicæmia results.

Septicæmia usually occurs from the inoculation of a wound, or of the uterus after delivery, etc., with the micrococci. In a few cases no wound can be found. Three conditions may result from the inoculation, namely, *septic intoxication*, *septic infection*, and *pyæmia*. The first tends to pass into the second, and the two last are so frequently associated that the name of *septico-pyæmia* is often applied to the combined conditions.

Pathology.—In **Septic Intoxication**, which is also called *sapræmia*, there are no cocci in the blood, but only the toxins which have been manufactured by the cocci (see p. 6). For instance, a piece of placenta left in the uterus may be full of cocci and their toxins, but the latter may be absorbed into the blood without the former. Also, cocci may live for some time in a septic wound without entering the blood, while their toxins are being freely absorbed. After death in these cases, blood-staining of the endothelium of the heart, pleura, etc., is found, and the organs, particularly the lungs and spleen, are soft and congested.

In **Septic Infection** the cocci are in the blood, and manufacture their toxins there. The effect is inflammation. At first the condition may be localized; for instance, if the site of inoculation be the hand, inflammation may spread up the lymphatics of the

arm, and cause inflammation of the glands at the elbow and in the axilla successively, before the blood is infected. Such cases are more favourable, as the infection may stop at any point. In other cases the blood is infected at once, so that the poison reaches the whole body. In either case, when the blood is infected, the serous membranes, and sometimes the mucous membranes, become inflamed. After death, the pleuræ, pericardium, etc., may be filled with fibrin and serum, and there may be small petechial hæmorrhages under the serous and mucous membranes, while microscopically the capillaries may be plugged with masses of micrococci. The appearances found in septic intoxication are also present.

In a very short time the septic infection leads to suppuration, and then the condition is called **Pyæmia**. The pleura, pericardium, etc., become filled with pus. Suppurative arthritis is also common. Very often, too, the blood coagulates in the veins next to the site of infection (*e.g.*, in the iliac veins in puerperal cases, and in the portal vein when the infection comes from an intestinal ulcer, etc.), and the resulting thrombi soften and suppurate, whence the name pyæmia (pus in the blood). Small infective particles then become detached from the thrombi, and are carried to the lungs, liver, etc., plug the bloodvessels in these organs, and become the foci of rapidly developing abscesses. If the particles detached be larger, they plug the arterioles and cause infarctions, which soon break down to pus. Cocci may also pass through the capillaries of the lungs, and become impacted in the brain, kidneys, etc., and set up abscesses there.

Symptoms.—In Septic Intoxication there is fever,

with headache, and perhaps even delirium. Diarrhœa is common, and bronchitis may occur. The temperature rises suddenly, and is very irregular in its course, because it falls as the toxins in the blood become less, and rises again when a fresh absorption of them occurs. Some cases die of the intoxication only.

When Septic Infection supervenes, various local lesions occur. In superficial parts, such as the arm or leg, red lines caused by lymphangitis may be seen on the skin, and the glands are painful and tender, and may suppurate. The temperature is high, but shows a distinct remission every twenty-four hours, and considerable sweating may occur while it is falling. Then the symptoms and physical signs of pleurisy, or of peritonitis, etc., may become present. If the patient does not die in this stage, pyæmia sets in. Suppuration then occurs in the pleura, etc. The joints become painful, red, and swollen, and are found to be full of pus. If a superficial vein, such as the femoral, be thrombosed, it becomes a painful, tender, solid cord, and œdema of the leg follows. Lastly, the symptoms and physical signs of abscesses in the lungs, liver, muscles, etc., may supervene. In the suppurative stage the temperature is very remittent, or even intermittent, and repeated rigors and sweatings may occur.

The septic affections most often met with in medical practice are those which arise from old ear disease, and infect the lungs first; those which arise from intestinal ulcers, and infect the liver first; and ulcerative endocarditis. In the last case the site of infection is often obscure, and the brain, spleen, and kidneys are usually the seat of suppurating infarctions.

Treatment.—The site of infection must be made as aseptic as possible by free opening and frequent washing out with antiseptic solutions. This is usually sufficient to cure septic intoxication. Even after infection has occurred this local treatment must be continued. For the rest, the fever may be treated with quinine, phenacetin, etc., and the heart with stimulants, and local lesions must be treated as they arise. An antitoxin prepared from cultivations of the streptococcus is being tried.

YELLOW FEVER.

Cause.—This is an acute specific disease, due to some unknown virus. It occurs in the tropical parts of America, particularly in hot low-lying districts. It, as a rule, prevails in epidemics, but is still endemic in certain seaports. The arrival of an infected person in a healthy district may occasion an epidemic, but it is not thought that there is any danger in being near a person who is ill with the disease.

The incubation period is from two to fourteen days.

Symptoms.—The onset is acute, with *high fever*, and all the febrile symptoms, particularly a very dry skin. There is intense headache and pain in the back; the *epigastrium* is painful and tender, and vomiting is common.

In about two days the fever and headache subside, but the epigastric tenderness persists, and *jaundice* is likely to occur. The skin becomes so intensely yellow that it is probably stained with hæmoglobin as well as with bile. Then the characteristic hæmatemesis, or *black vomit*, sets in, and there may be melæna and subcutaneous hæmorrhages. Albuminuria is also common, and the

symptoms of the typhoid state may develop. The disease is very fatal, and death may occur from exhaustion or from collapse, with or without hæmorrhage. The yellow colour of the skin may persist for a long time after recovery.

After death the *liver* is found to be large, soft, and yellowish, and the gastro-intestinal tract is somewhat inflamed, and may be studded with petechial hæmorrhages.

Treatment.—It is thought that calomel and quinine in large doses are useful if they are given at the beginning of the disease. For the rest the treatment is expectant, and in particular the fever, if high, should be treated by cold bathing.

MALARIA.

Cause.—The name of malaria was originally given to the noxious atmosphere which was known to cause certain diseases, such as intermittent fever, enlarged spleen, etc., and afterwards it came to mean the virus which was contained in that atmosphere. More recently the virus has been shown to be a parasite.

Malaria is *endemic*; *i.e.*, it exists in certain localities. These localities are most often situated in hot climates, and they are usually low-lying, swampy, and covered with vegetation. Apparently, the poison is present in the soil, from which it rises in a mist, or as an invisible exhalation, which is called a *miasm*. The atmosphere is most poisonous when the temperature of the ground is changing, *i.e.*, at sunrise and sunset, and also when the ground has been soaked by rain or floods, and is then baked by a hot sun. Digging up the ground liberates the

poison, and is a great cause of outbreaks of fever, but thorough drainage will often free a district of malaria altogether.

Other points about the malarial poison are that it does not rise very high from the ground, that it is absorbed by water, that it is carried by winds, and that it does not penetrate foliage.

The parasite is a protozoon, and is usually called the *Plasmodium malariae*. Other names for it are the *hæmatozoon* or *Hæm-amœba malariae*, because it is found in the blood. It was discovered by Laveran. It lives in the soil or in water, and enters the human body by the lungs, or by the alimentary canal in drinking-water. It is taken up from human blood by the mosquito, and on the death of that insect, it may reinfect the soil or water.

In the human blood the parasite is at first very minute, and it enters the red blood corpuscles. It then increases in size and distends the corpuscles. As it grows, it nearly always is found to contain pigment granules, which are derived from the hæmoglobin. At any time it may become free, from destruction of the corpuscles, and it may then be a more or less rounded, motile, pigmented body, to which certain thin, long, and flexible processes, called *flagellæ*, may be attached. Another curious form which may be found is a crescent-shaped body, which may also be pigmented. Finally, as the parasite matures, it becomes segmented, and then breaks up, each segment becoming a minute free parasite, which enters a red blood corpuscle, in which it undergoes the changes described above.

After death the parasite and various pigmented bodies may be found in the bloodvessels. The spleen is always enlarged, and in old cases it becomes

very large and firm and dark, while its fibrous stroma is much increased in quantity and highly pigmented. Such a spleen is called the *ague-cake*. The liver may show an intercellular cirrhosis, and the liver-cells and the endothelium of the capillaries may be pigmented.

Symptoms.—The affections produced by malaria are: (1) Intermittent fever or ague; (2) remittent fever; (3) enlarged spleen; (4) neuralgia; (5) anaemia.

1. **Intermittent Fever—Ague.**—This is the most typical affection produced by malaria, and it is characterized by attacks of fever which last a variable number of hours, and tend to recur at fixed intervals. Each attack can be divided into the following stages:

(a) *The Cold Stage.*—The patient suddenly or gradually feels cold, and has a rigor with shuddering and chattering of the teeth. Vomiting is a common symptom, and a large quantity of pale urine of low specific gravity is passed. There is also headache and pains in the back and limbs, and in spite of the feeling of cold, the temperature, if taken in the mouth, is found to be rising. The pulse is rapid and small, and the skin is pale or blue, and contracted, so that it is rough, like the skin of a goose (*cutis anserina*). This stage lasts from half an hour to an hour.

(b) *The Hot Stage.*—The patient at first feels warm, and then intensely hot, the temperature rising to 104° , or higher. The skin is dry and burning, and the cheeks are flushed. The pain in the head, back, and limbs is now intense, and any urine passed is scanty, high-coloured, and of high specific gravity. The pulse is rapid, full and

bounding, and there is great thirst. This stage may last from three to eight hours, and if it be prolonged, there may be delirium or coma.

(c) *The Sweating Stage*.—Perspiration sets in gradually, and soon becomes profuse. The temperature falls to normal, and the pains cease, but great prostration is left.

In bad cases jaundice may occur, and there may be hæmorrhages from the mucous membrane, or under the skin. The urine may contain hæmoglobin.

Varieties.—In *quotidian* ague an attack of fever like the one described above occurs every day; in *tertian* ague it occurs every other day; and in *quartan* ague every fourth day. In typical cases the attack comes on at the same hour on each appointed day. In *double tertian* ague an attack occurs every day; but the attacks on alternate days correspond with each other. For instance, the attack on Wednesday occurs at the same hour and has the same characteristics, as regards the length of the three stages, etc., as that on Monday, while the attack on Thursday corresponds to that on Tuesday. In the same way a *double quartan* ague and many other varieties may be recognised.

It is maintained that the attack of fever occurs when the segments of the parasite (see above) become free and invade fresh blood-corpuscles, and that quotidian ague is caused by a parasite which undergoes segmentation twenty-four hours after it has entered the corpuscles; while tertian ague is caused by one which takes forty-eight hours, and quartan ague by one which takes seventy-two hours, to undergo that process.

2. **Remittent Fever.**—There are two types of remittent fever. In one, the temperature varies from 99° to 101° in the twenty-four hours, and the febrile symptoms are comparatively mild. While the temperature is low, the patient may be able to go about, but feels wretched. As the temperature rises, there may be shivering and headache, and he usually has to lie down. Such a fever may last for weeks, and leads to great debility.

In the other type of remittent fever, the temperature varies from 102° to 104° in the twenty-four hours, and the patient is desperately ill. As a rule, he gets into the typhoid state, with a dry, brown, tremulous tongue, delirium, coma, etc. Jaundice and hæmorrhages are very likely to occur, and, unless the fever can be arrested, death will take place in a few days. The spleen, of course, gets large.

It must not be supposed that these two types are distinct varieties, as all kinds of intermediate types are met with, and the mild type may develop into the severe one, and the severe one may, after a few days, become milder, and then last for a long period, often until the patient leaves the district.

3. **Enlarged Spleen.**—The spleen increases in size during both intermittent and remittent fever, and often remains permanently enlarged. It may also become large without any definite attack of fever. It causes pain and inconvenience by its mere weight, and it has been known to rupture on very slight violence. In the early days of its enlargement it is painful and tender.

4. **Neuralgia.**—This is often called *brow-ague*, because it affects the supra-orbital branch of the fifth nerve, and because it is *periodic*, like ague; *i.e.*, the pain in the head may come on at the same

hour every day, or every other day, last for a few hours, and then pass off. There is tenderness over the nerve, and there may be slight shivering when the pain comes on. Intercostal neuralgia may also occur.

5. **Anæmia.**—This is a common result of malarial poisoning, and it is often associated with emaciation and great weakness, constituting a regular cachexia. It may occur after the fevers, or without any fever. The blood is deficient in red blood-corpuscles (oligocythæmia), and the white blood-corpuscles are increased in number (leucocytosis), particularly when the spleen is large, and they may contain black pigment granules (melanæmia). There is, often, hypochondriasis in these cases.

Persons who have resided in malarial districts are liable to attacks of fever and neuralgia even after they have resided out of those districts for many years. A chill or some very trivial cause may bring on the attack.

Treatment.—Quinine kills the parasite, and therefore cures the disease. In intermittent fever 10 or 20 grains of the sulphate of quinine should be given about three or four hours before the attack is expected; but 5 grains may be given three times a day in addition. During the attack, quinine usually causes vomiting, so the treatment must be palliative; *i.e.*, during the cold stage, the patient should be covered up with blankets, hot bottles should be placed in the bed, and hot drinks administered; while in the hot stage iced drinks should be given, and sponging with tepid water, or even a cold bath, may be useful. In the sweating stage, care should be taken to prevent a chill. In the intervals of the attacks, the patient should

take nourishing food and a moderate amount of stimulants.

In *remittent fever* quinine should be given in moderate doses three times a day. On the whole, this form of fever is less amenable to quinine than the other, and it may be necessary to try antipyretic drugs and the cold bath treatment.

In all cases of intractable fever, the patient should be sent away from the district.

In chronic malaria quinine must also be given. It often cures brow-ague at once, and will reduce the size of the spleen. Arsenic may be tried in all cases of malaria (even in the fevers), if quinine fails, but it is especially useful in chronic cases with anæmia. Iron is also useful.

The prophylaxis of malaria consists in taking quinine regularly during residence in a malarial district, or, at any rate, during the unhealthy seasons. For the rest, the evening and early morning air should be avoided, and only the upper rooms of a house should be used for sleeping in.

CEREBRO-SPINAL MENINGITIS.

Cause.—This is an acute specific disease, due to some unknown poison. It prevails in epidemics, and especially affects young males.

Symptoms.—The disease often sets in suddenly with rigors, or perhaps convulsions, vomiting, constipation, and headache. The temperature rises rapidly, but throughout the disease it runs a very irregular course. The symptoms are chiefly due to irritation of the cerebral and spinal nerves as they pass through the inflamed meninges. For

instance, contracted pupils and squinting are common symptoms, and result from irritation of the third nerve. Also neuralgic pains in the face, with an eruption of herpes, may result from implication of the fifth nerve, and convulsive movements of the features from implication of the seventh. At the same time, the neck becomes rigidly retracted, or there may even be opisthotonos, from irritation of the motor roots of the spinal nerves, while flashing pains round the waist or down the limbs may result from irritation of the sensory nerves. Later on the nerves which had first been irritated become destroyed, and then paralysis of the muscles which they supply sets in, and anæsthetic patches may be found in various parts of the skin.

The other symptoms of the disease are hæmorrhages, which are usually subcutaneous and petechial, but may take place from the mucous membranes, etc. Various symptoms of meningitis, such as a retracted abdomen, the *tâche cérébrale*, optic neuritis, etc., may develope, and finally the respiratory muscles may become paralyzed. Death in about a fortnight is the most common termination.

Post-mortem.—The brain and spinal cord are found to be covered with fibrin, and there is an excess of cerebro-spinal fluid. The ventricles of the brain may be distended as in tubercular meningitis, and the convolutions flattened.

The **Treatment** consists in counter-irritation, even by the actual cautery, over the spine, and the application of ice to the head. Chloral and bromides may be given to check the spasms, and it is wise to begin with a calomel purge.

HYDROPHOBIA—RABIES.

This is an acute specific disease, due to an unknown poison. The disease affects dogs chiefly, but may also occur in cats and wolves.

Human beings are usually infected by the bite of a rabid dog. This shows that the poison is present in the saliva, but Professor Pasteur has found that it exists in a concentrated form in the medulla oblongata of dogs which have died of the disease. Pasteur also found that the virulence of the poison could be modified by drying the medulla of a rabid dog in a bottle containing caustic potash; so that the longer it was dried, the less virulent it became. And, further, he found that if an animal were subjected to a series of inoculations with macerated medullas, beginning with the least virulent, and gradually going on to the strongest, no symptoms were produced in that animal, although rabies would certainly have followed if it had been inoculated with the strongest preparations without having been subjected to the complete series of milder ones in succession.

It would appear, then, that the series of inoculations protects the animal against hydrophobia, and, fortunately, the period of incubation of the disease in a man who has been bitten by a rabid dog is so long (varying from two or three weeks to several months) that there is time for him to be inoculated with the whole series of graduated viruses before the disease has matured in him, so that when it has matured, and should produce its effects, it finds him protected against it.

Symptoms.—The disease begins with restlessness and mental depression, with a feeling of causeless

fear. There is often a catch in the breath, or a sighing inspiration from irregular action of the diaphragm, and the chest feels oppressed. The subsequent symptoms are those of motor, sensory and mental excitability. There is a tendency to spasm of the muscles of deglutition, which first renders the swallowing of fluids impossible (hence the name hydrophobia), and then that of solids. The spasms are very painful, and they may extend from the muscles of deglutition to those of respiration. Any attempt to swallow, or even the sight or sound of fluids, or the mere idea of drinking, may occasion them, and they may eventually involve all the muscles of the body. The presence of saliva in the mouth is a great source of distress, because of the necessity of swallowing or expectorating it. Finally, any reflex stimulus, such as touching the skin, may set up the spasms. There is also great hyperæsthesia to light, sound, and touch. The mental condition may be melancholic and maniacal alternately, but there are often lucid intervals. Death may occur during a spasm of the respiratory muscles, but usually takes place gradually in the course of a few days from exhaustion.

Treatment.—Once the disease has become established, we can only try to give relief by prescribing chloral, bromides, etc., or by administering chloroform.

INFLUENZA.

This disease is often called *epidemic catarrh*, because it usually prevails in epidemics, and because the most common symptoms are those of conjunctival and nasal catarrh. But isolated

cases of the disease may be met with, and other and much graver symptoms than catarrh may occur.

Cause.—The disease is acute and infectious, and probably specific. A micrococcus which is considered to be the actual virus has been discovered; but the pathological conditions attributed to influenza are so many and so different one from the other that it is supposed by some authorities that, during an epidemic, the condition of the atmosphere is highly favourable to the development of all kinds of pathogenic bacteria, and at the same time highly depressing to the vitality of human beings, so that the latter are at the mercy of the former, each individual developing an affection in that part of his body which is naturally the weakest.

The outbreak of an epidemic in any locality can usually be traced to the advent of some person from an infected district; but in some cases this cannot be done, and it would appear as if the virus was borne by the winds. It has also occurred in ships that have been long at sea.

Symptoms.—A simple case of influenza begins with a sudden rigor, and perhaps vomiting. There is also headache and intense pain in the back and limbs, the temperature rises to 103° or more, and all the febrile symptoms set in. From the first there is great cardiac depression, the pulse, although rapid, being very weak and dicrotic, and sometimes intermittent. The eyes smart a good deal at first, and become suffused, while the nose and frontal sinuses feel stuffed. Then sneezing occurs frequently, and in a short time there is a copious muco-purulent discharge from the eyes and nose. The temperature falls to normal in the course of a few days, but

extreme weakness and depression delay the convalescence for perhaps several weeks.

In some cases there is severe sore throat, with *tonsillitis*. *Bronchitis*, with great cardiac depression, muscular pains, etc., may also occur, and may become capillary bronchitis or broncho-pneumonia. In some cases the bronchitis appears to begin in the capillary tubes. Ordinary croupous pneumonia is often epidemic at the same time as influenza; but in some genuine cases a *pneumonia*, with prune-juice sputum and very little dulness or tubular breathing, may occur, and be rapidly fatal. The pleura, pericardium, and endocardium sometimes become inflamed.

Influenza may also produce *gastro-enteritis*, with epigastric pain, vomiting, and diarrhoea; and in some cases nephritis is the prominent symptom.

In other cases, again, the *nervous system* is gravely affected, the headache being intense, and delirium or coma being likely to supervene.

A few cases begin with sudden extreme muscular *prostration* and cardiac failure, the seizure being as rapid as that of apoplexy. The nerves themselves are often affected, *neuralgia* being a common symptom, while paralysis from *peripheral neuritis* may occur as a sequela.

Lastly, mental failure, temporary or permanent, may follow influenza, and in old people it may bring on senile dementia.

Post-mortem.—If any lesions have occurred, they will, of course, be evident; but otherwise only such evidences of blood-poisoning as blood-staining of endothelial surfaces, and congestion with softening of the organs, will be found.

Treatment.—It is usual to give tinct. quiniæ

ammon., ʒi. , in water every four hours. In other respects the treatment is expectant—*i.e.*, quinine or salicylates must be given for high temperatures, stimulants for cardiac failure, etc., while bronchitis or gastro-enteritis must be treated in the usual manner.

SCURVY.

Cause.—This disease is due to the absence of fresh vegetables from the diet. It is consequently most common among sailors, but occasionally occurs among the poor on land. Bad air and bad food of all kinds may be considered to be auxiliary causes. The exact condition of blood which results from the deprivation of fresh vegetables is uncertain, but a deficiency of potassium salts has been suggested.

Symptoms.—The characteristic symptoms are those of progressive asthenia, with swelling of the gums and hæmorrhages. There is gradually increasing weakness, with aching of the limbs, dyspnœa on exertion, and a tendency to syncope. The patient is anæmic, and the complexion has a peculiar dirty yellow appearance. The amount of emaciation is very variable. There is no fever to speak of, but a great deal of mental depression.

The *gums* swell up enormously, project beyond the teeth, and bleed readily. The breath is very offensive, and in bad cases the teeth may fall out, and the jaw may become necrosed.

Hæmorrhages may occur under the skin, either in small purpuric spots or in ecchymoses, which change colour like a black and blue. Blood may also be effused deep down among the muscles of the limbs, particularly in the legs, where it forms large, painful, and tender swellings. At the same

time, bleeding from the nose, stomach, intestine, kidney, etc., may occur.

Treatment.—In the absence of fresh vegetables, scurvy can be prevented by the administration of an ounce of preserved lime or lemon juice daily. Vinegar is also said to be a preventive, but citric acid is of no use. When the disease is present, lime-juice (ʒiv. daily) will cure it. Fresh vegetables and fruit should be given freely. Tonics and stimulants are also useful. The mouth should be washed out with Condyl's fluid or boracic lotion, and for severe hæmorrhages tannic acid, ergot, and other astringents, may be tried. Recovery is usually rapid under treatment.

In **Infantile Scurvy** much the same symptoms occur, but, in particular, hæmorrhages frequently take place beneath the periosteum of the long bones, scapulæ, etc. They are very painful. Separation of the epiphysis from the shaft of the long bones is also common. The gums are not much affected until the teeth begin to appear. These cases occur in hand-fed children, particularly in those who are fed on artificially-prepared foods. They are easily cured by feeding on fresh milk, raw meat-juice, and potato soup.

PURPURA.

The name of purpura is given rather loosely to various affections in which small hæmorrhages occur under the skin. The hæmorrhages at first appear as pink spots, which soon become purple, and do not fade on pressure. They may be of any size, from that of a sixpence downwards, and they are not raised. If larger than a sixpence, they may

be called ecchymoses. As they get older, they change to green and yellow, like a black and blue.

Such purpuric spots occur in rheumatic fever (when they are called purpura or peliosis rheumatica), in typhus and other malignant fevers, in kidney disease, in certain cases of cirrhosis and of cancer of the liver, in pernicious anæmia, and leucocythæmia, and in other diseases in which the nutrition of the capillary walls becomes impaired. But in certain debilitated persons, who have no definite disease, but who have been ill fed and live under bad hygienic conditions, these hæmorrhages make their appearance, and it is to these cases that the name of purpura is applied, the one symptom giving its name to the disease. The spots are most commonly found on the legs, and there may be some fever. The disease can usually be cured in a week or two by good food, and tonics containing iron, arsenic, and bitters like gentian or calumba.

If the purpuric spots be accompanied by hæmorrhages from the mucous and serous membranes, or by subcutaneous ecchymoses, the disease is called purpura hæmorrhagica. Such cases may be accompanied by high fever and great prostration, and may even be fatal. They differ from scurvy in the fact that the gums are not spongy, although bleeding from them may occur. Further, purpura is not caused by the absence of vegetable food, and is not curable by lime-juice.

ACUTE RHEUMATISM—RHEUMATIC FEVER.

Cause.—This disease is hereditary, and the *pre-disposing* causes are cold and wet; but the *immediate* cause is some poison in the blood. Some

think that this poison is lactic acid, and that the effect of cold and wet is to disturb the metabolism of the organs, with the result that lactic acid is formed in excess, and so gets into the blood. Another view is that the poison is a micro-organism.

What is meant by the *nervous origin* of rheumatic fever is that the cold and wet act injuriously first on the nervous centres, and that these in turn upset the metabolism of the organs, with the result that lactic acid or some other abnormal substance accumulates in the blood. Another suggestion is that the arthritis is *trophic*, *i.e.*, that whatever the poison in the blood may be, it affects the nervous centres, and that the latter send some influence to the joints, which disturbs their nutrition.

Pathological Anatomy.—After death the *joints* are found to be filled with serum, in which some flakes of lymph (fibrin and leucocytes) may be floating. The cartilages look normal, but the synovial fringes are very vascular. Any complications, such as endo- or peri-carditis, will, of course, present their characteristic appearances.

Symptoms.—The disease begins with fever and pains in the joints.

The *joints* most frequently affected are the large joints, such as the knee, wrist, etc. They become painful and tender, and any movement causes extreme pain. Enlargement of the joint soon occurs from distension of its cavity with fluid, and fluctuation can usually be detected. As a rule, there is no thickening of the articular ends of the bones. There may be a pink flush where the joint touches the bed, etc., but there is no general redness, and suppuration practically never occurs. The joints may be affected together, or one after

the other, and the inflammation may subside in one joint while another is being attacked.

The *temperature* chart is very irregular, the temperature varying between 101° and 103° , but without regular daily remissions. The rate of the pulse and the other febrile symptoms are proportional to the temperature. In spite of the fever, the body is covered with a profuse, very acid *sweat*, and *sudamina* and *miliaria* are usually to be seen.

The duration of the disease is very uncertain, but it may last from a few days to several weeks. It is not dangerous in itself, but is often fatal through its complications. In a few cases the joints become permanently disabled from thickening of the articular ends of the bones.

Complications.—*Pericarditis* and *endocarditis* are common. The heart complications occur most often in children, and they lead to permanent valvular disease. The other serous membranes may also be affected, so that pleurisy may occur, and meningitis, or even peritonitis.

Hyperpyrexia occurs sometimes. The temperature may rise to 107° or higher, and then, as a rule, delirium and coma, and other symptoms of the typhoid state, set in, and death takes place in a few hours.

The other special complications are purpura, erythema nodosum, and simple erythema; but pneumonia and nephritis may occur, as in other fevers. Certain cases of iritis, tonsillitis, and appendicitis appear to be due to the rheumatic poison, while chorea and rheumatic fever are certainly associated diseases. Lastly, children who have had rheumatic fever sometimes develop small painful fibroid nodules in the skin.

The causes of death in rheumatic fever are pericarditis, endocarditis, and hyperpyrexia. For the Differential Diagnosis, see p. 106.

Treatment.—The joints may be wrapped in cotton wool, or fomentations, containing laudanum, may be applied to them. Internally, salicylate of soda must be given in 20 grain doses every two hours, provided that the patient can be seen frequently. The first effect of the salicylate is to relieve the pain in the joints; then it lowers the temperature and slows the pulse. Its administration should now be stopped, as, if it be continued, it causes buzzing in the ears, the pulse becomes weak and intermittent, and dangerous prostration may occur. Coma and albuminuria may also follow salicylate-poisoning. The urine of persons taking the salicylates gives a violet colour with perchloride of iron.

It would appear that the salicylate, although it stops the pain and fever, does not prevent the occurrence of endocarditis, and does not shorten the duration of the illness; *i.e.*, when the drug is stopped, the symptoms may return.

Failing the salicylate, bicarbonate of potash, or quinine, or antipyrin may be given. Hyperpyrexia must be treated by ice-packs or cold baths.

Patients who have had rheumatic fever are very likely to have it again, so that they should wear flannel underclothing, and avoid exposure to cold and wet.

SUBACUTE AND CHRONIC RHEUMATISM.

Cases are often met with in which the joints become, in the course of a day or two, swollen, painful, and filled with a moderate amount of fluid.

Both large and small joints may be affected, and there is some fever. Such cases may be called cases of subacute rheumatism, and they differ from true rheumatic fever in being less severe, in being less likely to affect the heart, and in being less curable by salicylates. They may last a long time, and are more likely to produce permanent joint affections than rheumatic fever.

Some cases, again, are still less acute, and are characterized by pain and slight swelling of the joints, but practically no fever. On the other hand, they do not present the symptoms of rheumatoid arthritis (see p. 100). These may be called cases of simple chronic rheumatism, and they may last an indefinitely long time. They may follow subacute rheumatism, or even rheumatic fever.

Treatment.—In all cases of articular rheumatism, it is right to give the salicylate of soda a fair trial. If it fail, the treatment recommended for rheumatoid arthritis should be tried.

GONORRHOËAL RHEUMATISM.

In the course of gonorrhœa, the joints—especially the knees—may become painful, swollen, and filled with fluid. The joints seldom suppurate, but have a tendency to become chronically swollen and painful. The affection is supposed to be due to the absorption into the blood of some toxic material from the urethra. The treatment is the same as that of rheumatoid arthritis.

MUSCULAR RHEUMATISM.

It is usual to regard certain painful affections of the muscles, which are excited by cold or wet, as

rheumatic in nature. In **Lumbago**, the lumbar muscles are affected in this way; in **Pleurodynia**, the intercostal muscles, and perhaps the diaphragm; and in **Torticollis**, or **Wryneck**, the sterno-mastoids. The affection may come on quite suddenly, and the affected muscles are always stiff. There is never any fever or other disturbance of the health.

In **Lumbago**, the patient may feel quite well, but on stooping he becomes unable to rise, on account of the pain in the back. Other cases come on gradually. The muscles are tender on pressure, but the vertebræ are normal.

In **Pleurodynia**, the pain prevents full inspiration, so that the disease may simulate pleurisy; but there is no fever, no cough, and no friction sounds can be heard. The muscles are, of course, tender.

In **Torticollis**, the neck may suddenly become twisted from painful rigidity of the sterno-mastoid.

Muscular rheumatism is best treated by warm applications and massage. As soon as voluntary movements can be performed, they should be encouraged. Internally, opium and salts of potassium should be given.

RHEUMATOID ARTHRITIS—RHEUMATIC GOUT.

Cause.—The exact cause is unknown, but cold and wet are predisposing causes. Some think that it is a trophic disease—*i.e.*, that the joints are affected through the action of the nervous centres. The reasons for this view are that the disease resembles the joint affections of locomotor ataxy (Charcot's knee, etc.), and that the muscular atrophy

appears in some cases to be greater than that which would follow disuse of the joint. The disease is more common in women and in elderly persons, but it may occur in the young. It is also more common among the poor. Laundresses, scrubbers, and gardeners, being exposed to cold and wet, are very liable to it.

Pathological Anatomy.—The cartilages of the joint become *larger* and *softer*, because the cartilage cells multiply; while the stroma becomes softer, and breaks up into fibrillæ. Then that part of the soft cartilage which is pressed upon by the opposing surface is worn away, until the bone on both sides is exposed. Then the two bone surfaces rub against each other, and, in consequence, undergo a *condensing osteitis*, the result of which is that they become hard, white, and polished like ivory (eburnation). The parts of soft swollen cartilage which are not exposed to pressure—*i.e.*, the parts at the sides of the joint—bulge outwards and form bosses, which ultimately ossify. At the same time, the synovial fringes become more vascular and larger, and they, too, may ossify and form osteophytes. The bosses and osteophytes greatly impair the mobility of the joint, and sometimes fibrous ankylosis takes place across the joint cavity.

Symptoms.—The disease may affect the large joints, especially the knee and hip, or the small joints, especially those of the hand. The *pain* is not acute, but of a constant aching type. It is very marked if the joint be kept in one position for a long time, so that it comes on in bed. It is also severe when the joint is first put in motion (whether voluntarily or passively), but passes off if the motion be continued. Excessive exercise, however, brings

it on again. It is soothed by warmth, and it is always worse in damp weather.

As regards the *knee*, in some cases it is first attacked acutely; *i.e.*, there is a little fever, and the joint becomes suddenly painful and distended with fluid. But even in acute cases the somewhat spongy swelling of the cartilages and the synovial membrane can be felt, and *creaking* on movement can be detected. Later on, and in cases which begin gradually, the thickening of the articular ends becomes very marked, and they are hard and bony, while the fluid is absorbed. After eburation, *grating* may be detected on movement. Eventually the joint may become practically immobile, and the muscles of the thigh waste considerably. Other large joints, such as the hip, elbow, and shoulder, may be affected in the same way.

In the *hand*, the thickening of the articular ends of the bones of the phalanges is early in evidence. The muscles on the ulnar side of the first phalanges are stronger and more often used than those on the radial side, so that the pressure on the softened cartilages is greater on that side. Consequently, the cartilage on the ulnar side wastes, while that on the radial side bulges, so that the fingers become deflected towards the ulnar side. This is the characteristic distortion; but, in addition, the first phalanges are usually flexed, the second phalanges extended, and the terminal ones flexed, so that the hand is like a claw, especially when the palmar muscles atrophy and cause a hollow in the palm.

The disease does not shorten life, but causes great and permanent crippling.

For the **Differential Diagnosis**, see p. 106.

Treatment.—Internally, cod-liver-oil, the syrup

of the phosphates, maltine, etc., should be given. Iodide and bicarbonate of potassium are also useful, and small doses of opium are soothing.

Locally, the joint should be kept warm with flannel. Warm douching, or soaking the joint in hot solutions of soda, gives relief. In some cases, painting the joint with iodine, or blistering it, are useful. Passive movements of the joints and massage of the muscles are to be recommended, except in the acute stages; and drinking the waters and taking the baths at various spas often produce the best results. In Britain, Bath, Buxton, Strathpeffer, etc., and abroad, Aix-les-Bains, Wiesbaden, Royat, etc., are good places of resort.

GOUT—PODAGRA.

Cause.—The morbid condition in gout is an excess of *uric acid in the blood*. This is called *lithæmia*. The blood being alkaline, the uric acid combines with an alkaline base in it, and forms quadrate of sodium, and eventually this urate of soda is deposited in the joints, which then become inflamed.

The tendency to gout is often *inherited*; but it may be acquired by over-indulgence in rich nitrogenous food, and by drinking rich wines, such as port, burgundy, and champagne, or malt liquors to excess. Deficient exercise is often associated with the over-feeding. Lead-poisoning also causes gout. The disease is most common in middle-aged males.

The way in which the above causes act so as to produce the lithæmia is uncertain. Some think that they upset the functions of the liver, so that all the nitrogenous food is not converted into urea, but only into the less oxydized uric acid; and as

uric acid is not very soluble, it is not easily eliminated by the kidneys, and therefore accumulates in the blood. Others think that the kidney is primarily at fault, and does not eliminate the ordinary quantity of uric acid, the result being the same as in the last case. Another view is that the derangement of metabolism which results in the lithæmia is brought about by the action of the nervous system.

Pathological Anatomy.—In the joints, the urate of soda is first deposited in the form of acicular crystals, which radiate from the capsules of the cartilage-cells near the surface of the joint. As the deposit increases, it appears as a white incrustation on the surface of the cartilage. Later on, it fills up the whole of the joint cavity, and then it infiltrates the ligaments of the joint. Finally, in the superficial joints, it accumulates under the skin, which often becomes ulcerated. These degrees of deposit may all be seen at a post-mortem, the joints which have only recently been affected showing the first condition, and those which were earliest affected the last.

At the post-mortem, also, the kidneys are nearly always found to be small, red, and granular, with white streaks of urate of soda in the pyramids. All the consequences of such a kidney, such as thickened arteries, a hypertrophied heart, serous inflammations, and even cerebral hæmorrhage, may be found.

Symptoms.—An attack of gout in the joints is often preceded by exhilaration of spirits and a sense of well-being, which is called the *euphoria*. In other cases, again, it is preceded by dyspepsia, headache, and a furred tongue, and the urine is loaded with urates. This condition may be called

the *dysphoria*. The attack may be excited by wet, by a slight injury to the foot, or by over-feeding.

The first attack nearly always occurs in the night-time, and affects the metatarso-phalangeal joint of the great toe. There is excruciating pain, and the joint becomes tender, swollen, hot, shiny, dark red, with distended veins coursing over it, and slightly œdematous. In bad cases, the whole foot and, if the thumb be affected, the whole hand may become red and œdematous. There is often, also, some fever, thirst, and anorexia. As a rule, the pain is less in the day, but returns again at night. The attack usually subsides in two or three days under treatment, and the skin over the joint desquamates.

With care, the patient may avoid a second attack; but in most cases the attacks recur, and many joints become successively affected. In an old case, the joints, especially those of the great toe and the fingers and thumbs, are distorted and fixed, because they are filled up with the urate of soda. In these joints, the white deposit may be visible through the skin, which gives them a piuk appearance, and over a few of them the skin may have ulcerated, so that the white matter is exposed. Such ulcers usually exude thin sero-pus.

The knee and other large joints may also be distorted and fixed, and in many cases deposits of urate of soda, called *tophi*, are found in the cartilages of the ears, eyelids, etc. Large deposits of urate of soda are often called *chalk stones*, because they look like chalk, and some of them do contain a minute quantity of lime salts.

There are various other symptoms attributed to gout, such as attacks of cardiac augia, gastralgia, rectalgia, asthma, vertigo, coma, etc. If the arthritis

should not be marked when any of the above symptoms are present, the gout is called *retrocedent*. The lithæmia may cause many of these symptoms, but most of them (*e.g.*, the asthma, coma, etc.) are due to the uræmia, which is caused by the granular kidneys which so often complicate gout. At any rate, deposits of urate of soda are rarely found except in connection with the cartilages.

Complications.—*Granular kidneys*, with their consequences, such as chronic endarteritis and endocarditis, a hypertrophied heart, asthma, uræmic coma, cerebral hæmorrhage, serous inflammations, etc.

Psoriasis and eczema are well-established complications, and *iritis* may occur.

The causes of death in gout are cerebral hæmorrhage, uræmia, aortic valvular disease, and serous inflammations, such as pericarditis. A few cases die of mere exhaustion from pain and confinement to the house.

For the **Differential Diagnosis**, see p. 106.

Treatment.—In a first attack, wrap the joint in a hot fomentation, with laudanum sprinkled on it, and prescribe: Vin. colchici, ℥ x.; tinct. opii, ℥ x.; magn. sulph., ʒi.; and aq. menth. pip. ad ʒi.

If there be heart disease, watch the effect of the colchicum carefully; and if there be kidney disease, do not give the opium.

For chronic cases, colchicum may be given, combined with the salts of lithia or any alkaline diuretics.

The diet should be chiefly farinaceous, and any meat which is allowed should be dressed plainly. Fatty and saccharine materials should be taken sparingly. Stimulants should be avoided; but if

they be necessary, hock, light claret, and gin are the best.

The spas best suited to gouty persons are Vichy, Carlsbad, Ems, Contrexéville, etc.

DIFFERENTIAL DIAGNOSIS OF ARTICULAR AFFECTIONS.

1. **Rheumatic Fever.**—(a) It resembles an acute attack of *rheumatoid arthritis*, because in both the knee and other large joints may become rapidly swollen, fluctuating and painful. But in rheumatic fever there are profuse sweatings, the temperature is high, and the ends of the bones are not thickened, while in rheumatoid arthritis the swelling of the bones of the joint can usually be made out, and there is little fever or sweating. Further, rheumatoid arthritis affects elderly rather than young people, and does not cause endocarditis.

(b) The resemblances between *gout* and rheumatic fever are merely that both cause arthritis. But the former attacks the small joints, and causes great redness and œdema, while the latter attacks the large joints, and causes fluctuation and little redness. The age and condition of the patient, the complications, and, in fact, nearly all the symptoms, are quite different in the two diseases.

(c) Rheumatic fever may resemble *pyæmia*, because in both a number of large joints become painful, swollen, and fluctuating, and in both there is fever and sweating. But in pyæmia the joints are more apt to be reddened, and the inflammation does not recede from one joint and go to another, as in rheumatic fever. Also, if the pyæmic joint be opened, pus will be found. Further, in pyæmia

rigors occur, and the sweating coincides with the remissions of the temperature, while in rheumatic fever the sweating is almost continuous, and the fever, although irregular, does not remit every day. Lastly, in pyæmia, the site of infection (a wound, etc.) may be found.

2. **Rheumatoid Arthritis.**—(a) An acute attack of this disease in the large joints may resemble *rheumatic fever* (see above).

(b) Rheumatoid arthritis of the hand resembles *gout*, because in both the phalangeal joints are inflexible, large, and distorted. But in gout there will be a history of an acute attack in the great toe, the pain will be greater, the distortion of the joint will be more symmetrical (radial and ulnar sides being more equally enlarged, so that the deflection to the ulnar side is less), and the urate of soda may be seen through the skin or in the ear, etc. In rheumatoid arthritis, an inspection of the knee will usually show great enlargement of the ends of the bones.

(c) Rheumatoid arthritis in the knee may resemble *Charcot's knee*, but in the latter other symptoms of locomotor ataxy will be found; the swelling of the joint is painless, while in rheumatoid arthritis it is painful. Lastly, the general tendency in Charcot's knee is to disintegration, with increased mobility of the joint, while in rheumatoid arthritis the mobility becomes decreased.

3. **Gout.**—(a) Gout in the hand resembles *rheumatoid arthritis* (see above).

(b) A *poisoned hand* may resemble gout, because in both there is great pain in the hand, with redness and œdema. But in gout moving the joints causes great pain, while in the poisoned hand

moving the joints causes little pain, but pressure on the palm of the hand causes pain by increasing the tension.

DIABETES MELLĪTUS.

Pathology.—1. In this disease the metabolism of the organs is profoundly disturbed, and, in particular, the organs concerned in regulating the supply of sugar to the blood are deranged, so that the blood contains much sugar, which is eliminated in large quantities by the urine.

2. *As regards the normal disposition of sugar in the body*, it is believed that all starchy materials taken into the body are converted into sugar, and carried as such to the liver by the portal vein. In the liver the sugar is converted into *glycogen*, and this glycogen is stored up in the liver and slowly reconverted into sugar by a ferment. Small quantities of the sugar are then carried off into the blood to be used up by the muscles, etc., as required. The relation of the pancreas to diabetes will be discussed below.

3. There are various theories as to what *alterations of these functions* are present in diabetes. Some think that the sugar sweeps through the liver into the hepatic vein without being stored up as glycogen at all. Others think that the ferment acts in excess, so that the glycogen is reconverted into sugar as soon as it is formed. Others think that the liver makes glycogen out of all kinds of food, even out of nitrogenous food, so that glycogen is present in excessive quantity. Also, it has been suggested that the organs do not take up the sugar in the blood, so that it accumulates there.

4. *As regards the cause of the alterations in function*, there is strong reason to believe that the primary derangement in diabetes is in the nervous system. The reasons are experimental and clinical.

(a) **Experimental.**—Puncture of the floor of the fourth ventricle causes glycosuria.

(b) **Clinical.**—In several cases of diabetes, tumours and other lesions of the medulla and other parts of the brain have been found. Also injuries to the head, and poisons which act on the brain, such as chloroform, may cause glycosuria. Again, diabetes is a hereditary disease, and occurs not only in the children of diabetics, but in the children of epileptics, dipsomaniacs, and other insane persons. Lastly, diabetes may appear in persons who have suffered great mental anxiety.

5. *As regards the pancreas*, cirrhosis or fatty degeneration of that organ has been found in certain cases of diabetes. But in other cases the pancreas is normal; and, further, cirrhosis of the pancreas has been found without any glycosuria. Experimentally, *complete* removal of the pancreas causes glycosuria, but if any portion of the gland be left, glycosuria does not follow, although the solar plexus, etc., has been as much injured as when the whole gland is removed. On the whole, it seems probable that the pancreas may supply some ingredient to the portal vein which is essential to the disposition of sugar in the body, so that in the absence of this ingredient the sugar accumulates in the blood.

Pathological Anatomy.—No constant lesion is found in diabetes. Tumours, etc., in the brain, and the pancreatic changes mentioned above, may be found in some cases, and lacunæ round the

vessels of the medulla have been described. The liver may be congested, and the kidney may show tubular nephritis.

Symptoms.—The patient may come complaining of weakness, of thirst, of polyuria, or of one of the complications. The disease is most common between thirty-five and forty-five.

The *urine* is pale, bright-looking, and acid. As many as 20 pints may be passed in the twenty-four hours, and the sugar (glucose) may amount to 50 grains per ounce. At the height of the disease, the amount of urea passed is increased, but towards the end it may be diminished. Even the sugar may disappear at the last, especially if there be fever. The specific gravity may be 1040, because of the sugar and the increase of urea. Albuminuria occurs, as a rule, before the end.

There is great *emaciation*, and the patient feels cold. The breath has a sweetish odour, the *thirst* is extreme, and in some cases the appetite is increased. The *tongue* is small, red, and glazed, and there may be constipation or diarrhoea. The *skin* is dry and very rough, and among complications *eczema* of the vulva or prepuce (with great pruritus), and *boils* and *carbuncles*, are common. Other complications are *gangrene*, which may follow trivial injuries; *phthisis*, which is very common; and *retinitis*, *optic neuritis*, and *cataract*. Headache is a common symptom, and mental or moral perversion occurs in some cases.

The weakness increases steadily, but the end is usually brought about by *diabetic coma*, which may come on after a slight intercurrent illness, or after fatigue or a slight injury. The patient feels headachy and giddy, and may vomit. There is

slight delirium, and then unconsciousness sets in. The breathing is rapid and deep, the air entering the lungs freely and noisily, but without any stertor, stridor, or wheezing. This kind of coma is attributed to the presence of acetone in the blood, and in many cases aceto-acetic acid is found in the urine. Other cases of diabetic coma resemble alcoholic poisoning, or uræmia and sometimes the unconsciousness is due to syncope. Coma is always fatal.

The duration of diabetes is very variable. With care life may be preserved for years, but any trivial disturbance of the health may bring on coma. In young persons the disease is more likely to be fatal in a short time.

It must be remembered that every case of glycosuria is not one of diabetes, for sugar may occur in the urine in gouty and other persons. If the glycosuria be intermittent, and disappear quickly on proper dieting, and if there be no emaciation and no grave complications, it may be presumed that the case is not one of true diabetes, especially if the patient be old.

Treatment.—There is no cure for diabetes. Opium or codeia ($\frac{1}{4}$ grain in a pill) may relieve the symptoms, and cod-liver-oil and tonics may be given to support the strength. A pancreatic extract causes improvement in some cases.

The diet is very important. All substances containing sugar or starch, such as sugar itself, jams, puddings, fruit, etc., and bread, biscuits, potatoes, cauliflower, peas, and all white vegetables, must be forbidden. Saccharin or glycerine may be used to sweeten tea or coffee, etc. As substitutes for bread, bran biscuits or almond rusks may be used.

Spinach, turnip-tops, lettuce, watercress, and other green stuffs may be taken, and meat of all kinds is permissible. Alcohol should be avoided, but, if necessary, hock, claret, or whisky must be given. Cold, exhaustion, and anything likely to bring on the coma, must be guarded against.

DIABETES INSIPIDUS.

This disease is characterized by the passage of a large quantity of urine which contains no sugar. Polyuria, due to a lardaceous or granular kidney, or to anæmia or other obvious disease, should be excluded, but there remain a certain number of cases in which 10 or 20 pints of urine may be passed per diem without any obvious cause. The patient is of a neurotic habit. The specific gravity is nearly always low, but in a few cases it is high.

In most cases there is thirst, and in some the general health is not greatly disturbed. But in a few cases, especially when the specific gravity is high, there is emaciation, prostration, and most of the symptoms of diabetes mellitus, but without the glycosuria. Such cases may be fatal. As regards treatment, tonics, especially iron and strychnia, should be given.

RACHITIS—RICKETS.]

Cause.—This disease begins in infants at about the period of dentition, *i.e.*, from the seventh month to the end of the second year. It is due to improper food, especially to starchy foods, like biscuits, etc., which the undeveloped salivary glands of children cannot deal with. It is not caused by syphilis.

Pathological Anatomy.—The disease affects chiefly the ossification of bones. In normal ossification the bloodvessels and osteoblasts grow from the previously-formed medullary spaces into the cartilage, which has proliferated in preparation for their reception. They should advance in an unbroken line and form new medullary spaces in the cartilage. At the same time, the old medullary spaces should be filled up by the adhesion of the osteoblasts to the trabeculæ, and the trabeculæ themselves should become calcified.

In rickets the cartilage-cells proliferate abundantly, but the osteoblasts and bloodvessels advance irregularly, pushing forward at one point and not at another, so that a piece of young bone may be found in the cartilage, while a piece of cartilage remains unaltered among the earlier-formed medullary spaces. Consequently a large mass of unfinished bone is found at the line of ossification. At the same time, the filling up of the medullary spaces and the calcification of the trabeculæ are delayed, so that the mass of young bone is soft and spongy.

Symptoms.—The special symptoms are often preceded by diarrhoea. The child may be thin or very fat, and it does not learn to walk. Sweating of the head occurs at night, and the child is hot, and throws off the bed-clothes. The spleen is enlarged, and the urine contains an excess of phosphates and lime salts.

The fontanelles do not close as they should, the teeth appear late, and the parietal, occipital, and frontal eminences (points of ossification) are large. Consequently the whole head appears large, the forehead in particular being prominent. The

junction of the ribs and costal cartilages is enlarged, giving each rib a beaded appearance, and the ends of the radius, tibia, and other long bones are also much enlarged. The affected bones are tender.

In bad cases deformities occur from bending of the softened bones. The ribs become flattened at the sides, so that the sternum is pushed forwards (pigeon-breast). The promontory of the sacrum is pushed forwards, and the tuberosities of the ischia pushed apart by the weight of the vertebræ, so that the pelvis becomes flattened. Lateral curvature of the spine may also occur. The femora become bent outwards and forwards, and there may be knock-knee or bowed knee. The tibia and fibula are bent outwards and forwards, but turn inwards rather sharply at their lower ends. If the child creeps about, the arms and clavicles may also be bent.

Laryngismus stridulus and spasms of the hands and feet occur in some cases.

The disease passes off after the third or fourth year, and the large spongy ends of the bones become properly ossified. If care be taken while the bones are soft, deformities do not occur, and the child may become very powerful.

Treatment.—The diet should consist of cow's milk with, later on, beef gravy, etc. As medicines, cod-liver-oil, lime-water, and the syrup of the phosphates, should be given; and sea-bathing is beneficial. The child should be kept on its back as much as possible, and it may be necessary to use splints to straighten the limbs.

SYPHILIS.

This is a specific contagious disease. It is acquired by sexual intercourse with an infected

person, or occasionally by the inoculation of a mucous membrane or the abraded skin with the discharge from a primary or secondary syphilitic lesion. It may be inherited from either parent.

Acquired Syphilis.—A papule appears at the site of inoculation in from three to six weeks, and soon ulcerates. The ulcer has a punched-out appearance, and its edges are hard. This is the *primary chancre*, and it is accompanied by non-suppurative enlargement of the inguinal lymphatic glands.

In a week or two the *secondary* stage sets in. There is a purplish roseolar rash on the chest, abdomen, and flexor surfaces of the limbs, and a sore throat due to general faucitis. Later on white patches, mucous tubercles, and ulcers occur all over the mouth and fauces, mucous tubercles occur on the genitals and anus, and various papular and scaly eruptions of a coppery colour may occur on the skin. Baldness (which is usually temporary) and iritis are common symptoms. The secondary stage of syphilis may go on imperceptibly into the *tertiary* stage, or there may be an interval of comparative health.

In the *tertiary* stage ulcers may occur on the tongue, lips, soft palate, larynx, rectum, skin, etc. Unlike the secondary ulcers, these cause great destruction of tissue (*e.g.*, the soft palate may be perforated), and may leave deep scars, which on the skin may be pigmented. On the skin, also pustular eruptions (leading to *rupia*) and palmar psoriasis may occur. The nails may be affected by onychia, or may become brittle and opaque. Periostitis, leading to nodes, or to abscesses, or even to necrosis of the bones, is not uncommon; and eye complications, such as keratitis and choroiditis, may occur.

But the great tertiary lesion is the *gumma*, which is a tumour composed of granulation tissue, infiltrating the part in which it grows. Gummata may appear under the skin, on the dura mater, in the liver, testes, and other organs. They usually caseate and become surrounded by fibrous tissue, but may soften and break down.

Syphilis is a great cause of chronic inflammation, and may produce sclerosis (leading to locomotor ataxy, bulbar paralysis, etc.), or endarteritis (leading to aneurism), or cirrhosis of the liver, etc. It also causes lardaceous disease.

Inherited Syphilis. — In this case there is no primary lesion. In the worst cases the fœtus dies and abortion occurs. In other cases the child goes to full term, but is born dead or dies in a few days, often with eruptions on the skin, and with chronic interstitial hepatitis and chronic interstitial pneumonia internally.

In most cases the child is born apparently healthy, but in about six weeks a dirty purulent discharge from the nose sets in, and the perinæum becomes covered with a coppery rash, which may ulcerate. The child then becomes extremely cachectic, the skin is dry, yellow, and inelastic, like that of an old person. The face is wrinkled, and the lips and chin show a coppery discolouration, due to the irritation of the nasal and buccal discharges. Ulcerations may occur in the mouth, particularly at the corners, where they may leave permanent scars; and mucous tubercles, rashes, etc., may appear as in secondary acquired syphilis.

Eventually subcutaneous and other gummata may form, and chronic inflammations, such as cirrhosis of the liver, may occur. The bones may

be affected and, in particular, the nasal bones may be necrosed, so that the bridge becomes greatly depressed. Nodes often occur on the cranial bones, where they form ridges which run parallel and close to the sutures. These nodes were described by Professor Parrot, and they are harder than rachitic thickenings, and are not confined to the parietal and frontal eminences. Thin parchment-like spots are also found in the cranial bones in congenital syphilis, especially in the occipital fossæ, and they are called *craniotabes*.

The primary teeth decay early, and the permanent teeth usually show a V-shaped notch on the free edge of the central incisors, or they may be peg-shaped. Chronic interstitial keratitis also is common, and gives the cornea a ground-glass appearance.

The power of transmitting syphilis to children becomes gradually exhausted, so that we often find that the first conception ends in abortion, the second in the birth of a dead child, and the third in the birth of a child that shows the typical symptoms in a few weeks. The fourth child may only show such late lesions as Parrot's nodes, and the later children may be quite healthy.

Treatment. — Mercury is the specific cure for syphilis. In adults the ung. hydrarg. may be rubbed in, or the liq. hydrarg. perchlor. and other preparations may be prescribed. For children the ung. hydrarg. should be spread on flannel and worn next the skin, or hydrarg. c. cret., gr. i., may be prescribed. Mercury must be given until the gums get sore, and care should be taken to prevent purgation. Mucous tubercles and condylomata disappear rapidly when dusted over with calomel.

For tertiary lesions iodide of potassium must be given, and the general health must be supported by cod-liver-oil, iron, etc.

TUBERCULOSIS.

Cause.—This is a specific disease. It is due to a thin, slightly curved, non-motile bacillus, which is shorter than the diameter of a red blood-disc.

The disease can be inoculated, and may be contracted by breathing an atmosphere laden with fresh bacilli, or by drinking milk from tubercular mammæ, etc. The bacilli only grow in persons whose tissues are a suitable soil for their development. Such persons are said to have the *tubercular diathesis*, and the diathesis may be transmitted from parents to children, or may be acquired by privation, intemperance, debilitating diseases, etc. In inherited cases the complexion is often brilliant, the skin delicate, the hair fair, and the eyes blue, with large pupils, but such a type is not invariable.

Pathological Anatomy.—Wherever the bacilli settle, a small tumour called a *tubercle* is formed round them. Microscopically this tumour consists of a multinuclear, multipolar giant cell in the centre, with epithelioid cells around it. Outside the epithelioid cells there are small round cells. The tubercle which we see with the naked eye is composed of a collection of two or three of such *giant-cell systems*. Once formed, the centre of a tubercle always caseates, and under favourable circumstances granulation tissue may grow round the cheesy portion, encapsule it with fibrous tissue, and prevent any further development. But in weak persons the tubercle softens and breaks

down, especially if pyogenic organisms gain access to it.

The term *scrofula*, which was originally applied to tubercular glands in the neck, and afterwards to tubercular ulcerations of the skin, is now disused.

Symptoms. — The effects of tuberculosis are usually more or less *local*; *i.e.*, the tubercles form in an organ and set up a subacute or chronic inflammation of that organ, which may become acute by being infected with pyogenic organisms. In this way tuberculosis causes pulmonary and laryngeal phthisis, bone disease, meningitis, ulceration of the intestines, lupus, Addison's disease, etc.

But sometimes the tuberculosis is *general*, *i.e.*, the bacilli get into the blood, and are deposited in all the organs at the same time. Consequently tubercles develop in all the organs; and as they are numerous, small, and discrete, they are called *miliary*. In some of these cases the blood is infected from an old tubercular deposit, *e.g.*, from pulmonary phthisis, bone disease, etc., and thus the diagnosis is easy. But in other cases the original seat of the disease is too obscure to be found clinically, and the symptoms are those of general blood infection only. Such a condition is called *acute general miliary tuberculosis*, or *tubercular fever*.

The symptoms may come on suddenly or gradually. The temperature is *remittent*, but varies much, fluctuating between 104° and 102° during some days, and between 102° and 100° during others. Sometimes there is a double remission each day. The pulse is not, as a rule, proportional to the temperature, but the other febrile symptoms (thirst, anorexia, etc.) occur as usual

Sweating takes place when the temperature is falling. The characteristic sign is the discovery of tubercles in the choroid coat of the eye by means of the ophthalmoscope. The physical signs of bronchitis may be caused by the discrete tubercles in the lungs; squinting, headache, constipation, and vomiting may be caused by tubercles in the meninges, and diarrhœa by tubercles in the intestines. The abdomen is usually flat, and the spleen is not very large. As a rule, the tubercles do not break down in time to furnish bacilli to the sputum or other discharge, but a family history of tuberculosis may help the diagnosis. The symptoms of the typhoid state (delirium, coma, dry brown tongue, etc.) come on gradually, and death usually occurs in from two to six weeks. In prolonged cases definite signs of tuberculosis may be found in various organs before death.

Treatment. — This is, at present, merely expectant. Koch's tuberculin has been unsuccessful, but attempts are being made to prepare a tubercular antitoxin.

CARCINOMA—SARCOMA.

These are specific diseases. The exact cause of them is unknown, but it has been suggested that both may be due to parasites which are of the nature of protozoa.

They both begin as a localized tumour, which increases in size, and grows into and replaces the normal tissue in which it is growing. They both also become infective; *i.e.*, secondary tumours, exactly like the primary one in structure, develop in the glands and organs in other parts of the body.

These tumours are called *malignant* because they infiltrate the organ in which they grow, because they reproduce themselves in other organs, because, if left alone, they increase steadily until death takes place, and because, if cut out, they tend to recur. The degree of malignancy is very variable in different kinds of these growths. They mostly cause local pain and tenderness, and lead to great emaciation and loss of strength.

The chief differences between carcinoma and sarcoma are as follows :

Carcinoma, or Cancer.—This occurs in elderly people. Microscopically the tumours consist of a fibrous stroma, in which are spaces called alveoli, and in the alveoli there are *epithelial* cells. They are of average malignancy, and they infect through the lymphatics, so that they reproduce themselves first in the lymphatic glands near the primary tumours.

Carcinomata grow from epithelial structures, such as the epidermis, mucous membranes, and glands.

Sarcoma.—This occurs in younger people, and microscopically the tumours are composed of *embryonic connective tissue cells*. The stroma is scanty, and is distributed among the cells, without forming alveoli. The vessels of sarcomata are very large and numerous, and have thin walls, and they often rupture. Some sarcomata are more infective than cancer, but others much less so. They infect through the veins, so that secondary tumours are apt to occur in the lungs.

Sarcomata grow from connective tissues, such as the skin, fasciæ, bones, etc.

Varieties of Carcinoma.—(1) **Scirrhus**, in which

the fibrous stroma is abundant and the alveoli small. (2) **Encephaloid cancer**, in which the fibrous stroma is scanty and the alveoli are close together. (3) **Squamous epithelioma**, in which the epithelium is squamous, like that of the epidermis, and grows in long finger-like processes into the fibrous tissue. At the end of the processes the epithelial cells may become *horny*, as on the skin, and be packed together concentrically to form *nests*. (4) **Columnar epithelioma**, in which the epithelium is columnar, like that of the intestines, and *lines* the alveoli, instead of filling them up. The alveoli look like large gland-ducts, the lumen being filled with mucoid or colloid matter. Colloid cancer merely means that the epithelium of a cancer of any variety has undergone colloid degeneration.

Varieties of Sarcoma.—These are classified, according to the shape of the embryonic cells, into : (1) **Spindle-shaped-celled sarcoma**, the cells of which may be large or small ; (2) **round-celled sarcoma**, the cells of which may also be large or small ; (3) **giant-celled or myeloid sarcoma**, in which large multinucleated cells lie in varying numbers in the midst of round or spindle-shaped cells. Sarcomata may contain newly-formed pieces of the tissue from which they grew, whence the names osteo-sarcoma, chondro-sarcoma, etc.

Both carcinomata and sarcomata may contain pigmented cells, and are then called *melanotic*.

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AIDS TO MEDICINE.

PART II.

DISEASES OF THE LARYNX, LUNGS, PLEURÆ,
PERICARDIUM, HEART, AND VESSELS. DISEASES
OF THE LYMPHATICS AND SPLEEN. BLOOD
AFFECTIONS. DISEASES OF THE THYROID
AND SUPRARENAL CAPSULE.

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PREFACE.

ANY attempt to include the essentials of a large subject within the compass of a small book necessitates the omission of many facts which seem important to the writer, and will doubtless be missed by the reader. Nevertheless, it is hoped that there is enough matter arranged in a convenient form in this book to make it a real 'Aid' to all those who are studying medicine.

NORMAN DALTON.

4, MANSFIELD STREET,
CAVENDISH SQUARE, W.
November, 1895.

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ADDISON'S DISEASE.

AIDS TO MEDICINE.

DISEASES OF THE ORGANS OF RESPIRATION.

GENERAL CONSIDERATIONS.

THE leading *symptoms* of diseases of the respiratory organs are : *Dyspnœa*, or shortness of breath, *cough*, and *expectoration*. *Pain* with or without *tenderness* is often present, and in many cases there is *fever*. Sometimes the other organs betray the presence of the lung affection.

The *physical signs* obtained by an examination of the chest are always of great importance.

Dyspnœa.

Causes.—These are too numerous to mention one by one, but they can be divided into two chief groups. In one the oxygen does not reach the blood in the capillaries of the air-vesicles, while in the other the blood does not take up the oxygen. Each of these groups must be subdivided.

1. **The Oxygen does not reach the Blood.**—This may be brought about by :

- (1) **The Want of Oxygen in the Inspired Air.**—This occurs when the atmosphere is charged with excess of carbonic acid, etc.

(2) **Obstruction of the Air-passages.**—The obstruction may be : (a) At the *mouth* or *nose*, as in the case of large tonsils and post-nasal adenoids, etc. ; (b) in the *larynx*, as in the case of the entrance of foreign bodies, or in diphtheria, cancer of the larynx, etc. ; (c) in the *trachea*, as in strangulation, the pressure of an aneurism, etc. ; (d) in the *bronchi*, as in asthma, excess of bronchitic secretion, etc. ; (e) in the *air-vesicles* themselves, when they are filled with fibrin, blood, etc., as in pneumonia, etc.

(3) **Non-expansion of the Air-vesicles.**—This may be due to : (a) *compression of the lung* by pleuritic or pericarditic effusion, tumours, etc. ; (b) *deficiency in the respiratory movements* of the chest, such as occurs when the muscles of respiration are paralyzed, or when respiratory movements cause pain, as in peritonitis, pleurisy, etc.

2. **The Blood does not take up the Oxygen, although the Oxygen has reached the Air-vesicles.**—This may be brought about by :

(1) **The Destruction of the Capillaries of the Lungs**, such as occurs in phthisis, gangrene of the lung, etc., and in emphysema.

(2) **The Absence of Blood from the Air-vesicles**, although the latter may be normal. This occurs in embolism of the pulmonary artery.

(3) **The Stagnation of the Blood in the Air-vesicles**, such as occurs in the mechanical congestion due to mitral disease. In this

instance the blood could take up oxygen, but as it cannot leave the lungs, the result to the rest of the body is the same as if it could not.

- (4) **The Improper Quality of the Blood**, which prevents it taking up enough oxygen. This occurs in anæmia, or when there is a poison in the blood, as in the fevers, diabetes, etc.

In all cases, of course, the immediate cause of the dyspnœa is the poisoning of the respiratory centre in the medulla by carbonic acid.

Symptoms.—Whenever the breathing averages more than 20 per minute, dyspnœa may be said to be present. In all severe cases the alæ nasi work, and there is *cyanosis*, i.e., blueness of the face, lips, and conjunctivæ. Inability to breathe when lying down is called *orthopnœa*. It is especially important to distinguish dyspnœa due to laryngeal obstruction from that due to diseases of the bronchi or lungs, because tracheotomy will relieve the former. Laryngeal dyspnœa is characterized by *stridulous breathing* (which means a loud whistling or hissing sound made both during inspiration and expiration), and by *sucking in of the lower ribs and episternal notch during inspiration*. The last phenomenon is due to the fact that when the diaphragm goes down the air cannot enter the lungs, and consequently the atmospheric pressure presses in the yielding parts of the chest-wall.

The **Treatment** of dyspnœa depends on the cause, but it must be remembered that in all cases blood containing an excess of carbonic acid has a difficulty in getting through the lungs. Consequently, in all cases, the *right ventricle* tends to become distended,

and if unrelieved will stop, paralyzed from over-distension. Hence, while the treatment of the cause of the dyspnœa is being adopted, it is necessary to keep the right ventricle going, and this can best be done by stimulants, such as brandy, ether, and ammonia, and cardiac tonics like digitalis and strychnine. In suitable cases, too, the right ventricle can be relieved by venesection, for if 8 oz. of blood be suddenly withdrawn from the arm, and therefore does not reach the ventricle, the muscle of the ventricle may have a chance of contracting fairly on its contents, and so the over-distension may be warded off for a time.

Opium must *never* be given during urgent dyspnœa, because in all such cases the patient must use his voluntary muscles of respiration as well as his involuntary. Now, opium, whether it induces sleep or not, blunts the sensibility, so that the patient does not feel the necessity of keeping up strong respiratory efforts. But as soon as these are relaxed fatal carbonic acid poisoning will occur. Opium, also, by diminishing the desire to cough, allows the secretion to accumulate in the lung, perhaps to a fatal extent. If, however, it is certain that dyspnœa is merely due to the pain of moving the chest-wall, as in pleurisy and rheumatism, opium, by making the movements painless, may remove the dyspnœa.

Cough.

Causes.—Cough may be due to :

1. **Direct Irritation** of the larynx, air-tubes, or air-vesicles by muco-pus, blood, etc.
2. **Reflex Irritation**, as when the pleura is irritated by a broken rib or by pleurisy, or when the gastric

branches of the vagus are irritated in dyspepsia, etc.

What is called a *throat* cough is usually due to direct irritation of the arytenoids by phlegm, which is hanging about the fauces or pharynx, or by an elongated uvula. As the phlegm is not in the line of passage of the expired air, it cannot be expelled by coughing, and therefore is difficult to dislodge. The process known as *hawking* is used to effect its dislodgment.

A *dry* cough is one which is not accompanied by expectoration. Reflex coughs are always dry, and in the early stage of many lung affections, particularly phthisis, the expectoration may be very scanty.

As regards the characteristics of different kinds of cough :

The **Throat** cough is loud, hacking, frequently repeated, and accompanied by tickling in the throat. The sputum is brought up with difficulty.

The **Laryngeal** cough is hoarse, or barking. It has been called croupy.

The cough in **Emphysema** is attended by a prolonged expiratory wheeze.

The other lung coughs are not characteristic, except, perhaps, the hollow cough with a rattle of crepitation at the end of it, which occurs in the last stage of phthisis. It is the true churchyard cough.

The cough in **Pleurisy** is short and dry. It is essentially a *stifled* or *suppressed* cough, the patient trying not to cough because of the pain it excites. The cough in **Diaphragmatic Pleurisy** or **Rheumatism** causes the patient to be doubled up, because he tries to cough without using the painful diaphragm.

The cough due to irritation of the **Stomach** is a

loud, reverberating cough, unattended by expectoration.

The **Treatment** of cough consists in trying to remove the cause. For instance, rhubarb and magnesia are useful for a stomach cough, and sedative lozenges for a throat cough. As a general rule, a *dry* cough may be treated with opiates, balsams, syrups, etc., to diminish the irritability of the mucous membrane; but when there is expectoration, these remedies should, as a rule, be avoided, as they tend to make the lung tolerate the presence of the sputum instead of ejecting it.

Expectoration.

The material expectorated is called *sputum*, or in the plural *sputa*.

The following are the characteristic kinds of sputum :

The **Rust-coloured** sputum of *croupous pneumonia*, which contains fibrin, red blood-discs, leucocytes, and pneumo-cocci. It is tough, and adheres to the spittoon.

The **Plum-juice-like** sputum of *bad cases of pneumonia*, which contains much blood.

The **Nummulated** sputum of *phthisis*, which contains leucocytes, epithelioid cells, elastic fibres, and bacilli. It consists of little circular flat masses like coins, which do not run together in the spittoon. It may be streaked with blood.

The **Red-currant-jelly-like** sputum of *cancer* of the lung, which contains mucus and blood and cancer-cells.

The **Black** sputum of *anthracosis* of the lung. It contains angular particles of coal-dust, and occurs in miners, who acquire bronchitis by constantly

inhaling coal-dust. Most dwellers in large cities expectorate a little mucus mixed with coal-dust in the morning.

The Pearly sputum of *asthma*, which contains (a) Curschmann's spirals, *i.e.*, spirals composed of mucus which has been inspissated to a ropy consistence, and (b) Charcot's crystals, which are minute, colourless, pointed octahedra.

The Gangrenous sputum of *gangrene* of the lung. It is very offensive, gray, and contains leucocytes, bacteria of various kinds, and elastic fibres and bloodvessels from the dead lung tissue.

The Plastic sputum of *diphtheria* or *plastic bronchitis*. This consists of a *fibrinous* cast of the trachea or bronchial tubes. If it is a complete cast of many tubes, it will be branched like a tree, the branching corresponding to that of the bronchi.

In perichondritis of the larynx pieces of dead cartilage may be expectorated, and in hydatids of the lung or liver the cysts may be seen in the sputum.

When the sputum is very abundant and thin, the condition is called *bronchorrhœa*. It is due to chronic bronchitis with dilated bronchi. If there be a bronchiectasis, *i.e.*, a cavity formed by a dilated tube, the sputum may be brought up in a large quantity at considerable intervals, and is very offensive.

The expectoration of blood is called *hæmoptysis*.

HÆMOPTYSIS.

The Causes of hæmoptysis are: *Phthisis*, in which the blood may come from the larynx or the lungs; *croupous pneumonia*; *cancer* or *sarcoma* of any part of the air-passages; extreme mechanical *congestion*

of the lungs, such as follows mitral disease; hæmorrhagic *infarction* of the lung; the bursting of an *aneurism* into any part of the air-passages; *laceration* of the lung, caused by violent coughing or a broken rib; vicarious *menstruation*; *general diseases*, such as scurvy, purpura, hæmophilia, malignant fevers, etc.

Symptoms.—The blood is expelled by coughing. It is bright red, frothy, and may be mixed with ordinary sputum. It continues to be coughed up in pellets, after the first gush has ceased, and in big hæmorrhages the appearance of the blood and the faintness due to its loss occur together. Lastly, there will be symptoms and physical signs present to indicate disease of the larynx or lungs. Fluid blood in the lungs causes crepitation.

In *hæmatemesis* the blood is expelled by vomiting. It is dark, and may be mixed with food. The faintness precedes the ejection of the blood, and the other symptoms and physical signs point to the stomach or liver.

Treatment.—Give ice to suck, and apply ice to the chest-wall. Internally give ergot, gallic acid, dilute sulphuric acid, and other astringents. Absolute rest is essential. Opium is the best drug to stop the bleeding; but it has the disadvantage of stopping the cough, and therefore the expectoration of the blood. And if the blood remains in the lung it may become septic, and so cause extensive pneumonia. Still, if the hæmorrhage threatens life, opium must be given.

Pain and Tenderness.

In most laryngeal affections there is pain on speaking, and tenderness over the larynx.

Bronchitis of the large tubes causes pain, because the fibro-cartilaginous walls of the tubes are unyielding, and therefore the inflammatory swelling causes great tension.

The lung itself is not very sensitive, and the pain that accompanies so many lung affections is due to the adjacent pleura becoming involved.

THE PHYSICAL SIGNS OF LUNG DISEASE.

These are ascertained by (1) Inspection, (2) Measurement, (3) Palpation, (4) Percussion, (5) Auscultation.

1. **Inspection.**—(a) Inspection of the *surface* of the chest may reveal an increase in size of one side, as in large pleuritic effusions; or a localized bulge, as in a pointing empyema; or a localized depression, such as often occurs over a large cavity. Epigastric pulsation of the heart may be seen, and is very important, as it usually means dilatation of the right ventricle, which is a common consequence of lung disease. Enlarged veins over the chest are also worth noting.

(b) Inspection of the *respiratory movements* of the chest is of great importance. *Normally* during inspiration all parts of the chest-wall should move equally, and should expand laterally while they are being drawn upwards. Inspiration should take longer than expiration. *In disease* there may be general deficiency of expansion, the chest being merely drawn up as in emphysema; or one part may not expand as well as the rest, as in pleurisy, phthisis, etc.; or expiration may take longer than inspiration, as in asthma and emphysema.

2. **Measurement** of the whole or part of the chest

is used to determine accurately the observations which have been made by inspection, or to detect differences in size which are too small to be observed by inspection.

3. **Palpation.**—(a) By *ordinary palpation* a local bulge can be examined, and œdema of the chest-wall, the separation or approximation of the ribs, or the bulging of the intercostal spaces, can be detected. Epigastric pulsation may, perhaps, be felt when it cannot be seen.

(b) *Palpation for Fremitus.*—This is performed by placing the palm of the hand lightly on the chest-wall. If this be done while the patient is speaking, a thrill, called *vocal fremitus*, is imparted to the hand. Vocal fremitus is usually increased when the lung is solid, as in pneumonia, and lost when fluid or air in the pleura separates the lung from the chest-wall.

Apart from speech, a vibration can be felt when the air passes through a bronchus which is partially blocked by tough muco-pus. This is called *rhonchal fremitus*.

It is also possible to feel the friction caused by the movements of a very rough pleura.

4. **Percussion.**—This is performed by placing the forefinger of one hand flat on the chest-wall, and striking it smartly with one or two fingers of the other hand, in a direction at right angles to the chest-wall. Sometimes a kind of hammer, called a plexor, is used to strike with instead of the fingers; and an ivory plate, called a pleximeter, can be placed on the chest as a substitute for the finger which is struck. The finger, however, is the best pleximeter, as it appreciates the amount of the resistance of the chest-wall to the blow, and, in cases of solidifica-

tion of the lung, this is almost as important as the dull sound which results from the stroke.

The lungs, being filled with air, are resonant on percussion. Pulmonary resonance can be elicited for about one inch above each clavicle, and over the whole of the thorax except where the heart and liver touch the chest-wall. For the area of præcordial dulness see p. 62. The liver dulness extends on the right side from the base of the ensiform cartilage through the fifth and seventh intercostal spaces to the eleventh rib at the back. At the edge of both cardiac and hepatic areas there is a zone of about half an inch, over which there is partial dulness because the lungs overlap the solid organs. On deep inspiration the expanded lungs still further diminish the dull areas.

In disease the note on percussion may be *hyper-resonant*. This occurs when the lung is overdistended with air, as in emphysema.

Or the note may be *comparatively dull*. This occurs when small solid areas of tubercle, lobular pneumonia, etc., are present in the lung, but are separated from each other by normal areas containing air.

Or the note may be *absolutely dull*. This occurs : (a) When large areas of the lung become solid from pneumonia, tubercle, collapse, tumours, etc. ; (b) when the lung is separated from the chest-wall by fluid in the pleura or pericardium ; (c) when tumours of the mediastinum (*e.g.*, cancerous glands, aneurisms, etc.) or the liver, etc., grow between the lung and the chest-wall.

In addition to the above, there is the *cracked-pot* sound, which is a high-pitched note with a break in it. To elicit it, the patient should keep his

mouth open during percussion. It is best obtained over a large cavity, the wall of which consists of a thin strip of solid lung, adherent to the chest-wall.

Lastly, a high-pitched note, resembling the last, but without a break in it, is sometimes obtained on percussion. It is like the sound produced by tapping an empty box made of thin wood. It may be elicited (*a*) over a cavity; (*b*) over a piece of lung, the air-vesicles of which are so distended with air as to be stretched to the utmost; (*c*) in some cases of pneumo-thorax, when the air in the pleura is under very high pressure. In this last case, the note may be so high-pitched as to be almost inaudible, whereby it simulates a dull note.

5. **Auscultation.**—For this purpose a stethoscope is used, or the ear may be applied directly to the chest-wall. The breath-sounds and the voice-sounds must be auscultated.

(*a*) *Auscultation of the Breath-sounds.*—(1) The breath-sounds may be *normal*. Normal breathing is called vesicular, because the sound is made by the air rushing into the air-vesicles. The inspiratory sound should be longer and louder than the expiratory. Loud vesicular breathing is called *puerile*, because the breath-sounds are always loud in children.

(2) The breath-sounds may be *feeble* or *absent*. This occurs when the larynx is blocked; or when a pleuritic effusion compresses the air-vesicles and separates them from the chest-wall; etc.

(3) The *expiratory* sound may be *prolonged*, so as to be equal to or longer than the inspiratory, as in emphysema and asthma.

(4) The breath-sounds may be *tubular* or *bronchial*. These terms should be synonymous, because the tubes are the bronchi. In perfect tubular breathing the inspiratory and expiratory sounds are equal in length, and both have a peculiar hollow character which, to be appreciated, must be heard.

Now, it must be clearly understood that tubular breathing is the sound made by the air as it passes to and fro through normal air-tubes. In a normal person we cannot hear this sound when we listen over the *lungs*, because it is completely masked by the noise made by the air as it rushes into the air-vesicles—*i.e.*, by the normal vesicular breathing. But when the air-vesicles become filled with solid material, or are compressed so as to form a solid mass, this vesicular sound ceases; and as solid matter conducts sound well, we now hear the air moving in the tubes with great distinctness; *i.e.*, we hear tubular breathing. Hence tubular breathing nearly always means that the lung is solid. It does not matter what the cause of the solidification may be, whether it be caused by pneumonia, phthisis, collapse, sarcoma, etc.; so long as the tubes are open and the solid part touches the chest-wall, tubular breathing will be heard.

In some cases the tubular breathing may be imperfect; for instance, we may notice that the expiration has a tubular character, but not the inspiration.

(5) The breath-sounds may be *cavernous* or *amphoric*. This is simply very loud tubular breathing, and indicates a cavity, a dilated bronchial tube or a pneumo-thorax.

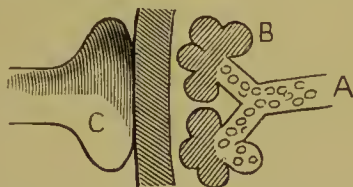
(6) *Râles* may be heard. A *râle* is an *abnormal* sound made by the air as it passes through the air-tubes or into the air-vesicles, and

practically râles always mean that there is some obstruction to the passage of air. They are divided into *dry* râles and *moist* râles.

Dry râles are heard when the tubes are narrowed by the contraction of their walls (as in asthma) or by the presence in them of a secretion which is too thick for the air to penetrate. Dry râles are subdivided into large and small, the larger being called *rhonchi*, the smaller *sibilli*. When a large tube is only partially blocked by a tough mass of mucus, the air passing over this mass causes a vibration which produces a snoring sound, or *rhonchus*. A similar tough mass of mucus, if situated in a small tube, leaves only a small chink for the air to pass through, and then the vibration produces a squeaking or whistling sound called a *sibillus*. Rhonchi and sibilli often disappear or change their position when the patient coughs, the cough dislodging the obstructing pellet.

Moist râles occur when the material in the tube is sufficiently thin to allow the air to bubble through it, and they are synonymous with *crepitation*. Crepitation is also divided into large and small. The former is produced by the bubbling of air in fluid in large tubes or cavities, and the latter by the fine bubbling in the small tubes or air-vesicles. Fine crepitation may also be produced in another way, as follows: When the lining of the air-vesicles becomes sticky (as in the earliest stage of croupous pneumonia), the opposing walls of each vesicle adhere and prevent the air entering during an ordinary inspiration; but if a deep inspiration be taken, the air forcibly separates the walls and enters the vesicles, making a succession of little clicks which sound together like fine crepitation.

Lastly, either large or fine crepitation may be *consonating* — *i.e.*, it may have a peculiar hard rattling character, differing from simple crepitation. This quality of consonating is added to crepitation when the latter is heard through solid lung. For instance, suppose that ordinary crepitation is being produced in the tube A by the bubbling of air through the fluid in the tube. Then if the lobule B becomes solid from any cause, this crepitation is heard through the stethoscope C more loudly and with a harsher character, and it is then called *consonating crepitation*.



Such terms as *mucous râles* and *mucous crepitation* should be avoided, as they imply that the fluid in the tubes is mucus, of which there is no evidence. When the sounds heard are suggestive of crepitation, but are not quite characteristic, it is usual to say that doubtful crepitation is present, or to call the sound a sub-crepitant râle.

(7) *Metallic tinkling* may be heard on auscultation. Such a sound is usually produced by a mixture of air and fluid in a large cavity, or in a pyo-pneumothorax, and it is not very important.

Lastly, when the surface of the pleura is roughened, a *friction-sound* is heard as its two layers rub together during respiration. There is usually an inspiratory and an expiratory rub, called therefore *to and fro*.

In many cases the sound is dry or creaking and superficial, and it is then quite characteristic; but in other cases it closely resembles fine crepitation. The latter, however, is usually only inspiratory, and the presence of pain on inspiration will help us to decide that the sound is friction, and not crepitation.

(b) *Auscultation of the Voice-sounds*.—The sound heard through the stethoscope when the patient speaks is called *vocal resonance*, and it may be normal; or increased in intensity; or diminished; or even inaudible, as in large pleuritic effusions. When the lung is solid, the vocal resonance has a characteristic sound, which is called *bronchophony*. Bronchophony may be louder or less loud than normal vocal resonance; but it has a quality of its own, the sound of the voice being conducted up to the ear in a peculiarly sharp manner. If we could place the stethoscope on a normal bronchus in a normal lung when the patient was speaking, we should always hear bronchophony; but the intervention of the air-vesicles containing air alters the sound to normal vocal resonance. When, however, the air-vesicles become solid, we hear this sound most distinctly. Hence bronchophony, like tubular breathing, indicates that the lung is solid, and the two are nearly always associated.

Sometimes bronchophony is extraordinarily well marked, and the sound passes up to the ear in the most striking manner, especially if the patient whispers, and it is then called *pectoriloquy*.

If the bronchophony is very loud and reverberating, it is called *cavernous*, or sometimes *amphoric*, vocal resonance, since the sound resembles that made on speaking into the mouth of an empty

jar (*amphora*). Such voice-sounds are heard over large cavities or a pneumo-thorax.

Lastly, the voice has sometimes a brassy sound, like the bleating of a goat, and it is then called *ægophony*. This is usually heard at the upper level of a pleuritic effusion.

Another method of investigating the chest is to place the ear directly on the chest-wall, and shake the patient's shoulders gently. Such shaking is called *succussion*. If there be a mixture of air and fluid in the pleura or in a large cavity, a splashing sound will be heard on succussion. It must be remembered that fluid alone cannot splash; there must be air mixed with it.

An examination of the heart is an essential part of an examination of the lungs. This especially applies to the extent of præcordial dulness, which is diminished in emphysema; to the position of the apex-beat, which is displaced in pleuritic effusion, etc.; and to the size of the right ventricle, which becomes hypertrophied, and afterwards dilated, in all chronic lung affections. An accentuation of the second sound at the pulmonary valve is also common in chronic lung affections, because the tension in the pulmonary artery becomes increased.

DISEASES OF THE LARYNX.

INFLAMMATION OF THE LARYNX— LARYNGITIS.

Acute Laryngitis.

Causes.—Cold and damp ; over-use of the voice ; the inhalation of steam or acid vapours, etc. ; the specific fevers, especially measles, whooping-cough, typhoid, etc. Diphtheria also causes a most intense laryngitis, but of the membranous variety (see p. 24).

Pathological Anatomy.—The mucous membrane becomes red and swollen. It is at first dry, but soon there is a secretion of muco-pus.

Symptoms and Physical Signs.—The *voice* becomes hoarse, and may be quite lost (*aphonia*). There is a peculiar, hoarse, barking *cough*, with, after a time, muco-purulent sputum. There is *pain* in the larynx, especially on speaking and swallowing ; and there is *tenderness* when the larynx is compressed from without. There is also slight *fever*.

With the laryngoscope, the mucous membrane is seen to be red and swollen. The swelling particularly affects the ary-epiglottidean folds, which project and obscure the view of the true cords. The latter, if seen, are not much swollen, and only slightly reddened. Normally, they look white and glistening. A good deal of secretion may also be seen lying on the surface of the mucous membrane.

In all laryngeal affections (except paralysis), the larynx may become obstructed, sometimes quite suddenly. Hence the symptoms of laryngeal obstruction must be mentioned as likely to occur in acute laryngitis. The obstruction, in acute cases, may be due to (1) inflammatory œdema of the loose submucous connective tissue, or (2) spasm of the adductor muscles.

Symptoms of Laryngeal Obstruction.—These are : aphonia ; dyspnœa, with *stridulous* breathing, in which a loud, whistling sound is made with both inspiration and expiration ; sucking-in of the lower ribs and episternal notch during inspiration (see p. 3) ; deficient entrance of air into the lungs, discoverable by auscultation ; cyanosis ; a small, rapid pulse, with, for a time, violent action of the heart.

If the obstruction be due to spasm, the symptoms come on suddenly, and may intermit from time to time, when the spasm relaxes. If due to œdema, there are no intermissions.

Treatment.—Apply poultices or compresses externally to the larynx. Adults should be made to inhale steam, and in the case of children a steam tent should be rigged up over the bed. Internally, ipecacuauba, vinum antimoniale, and carbonate of ammonia, are useful, and aconite may be tried. If there be much spasm, belladonna, bromides, and chloral, are useful ; and if laryngeal obstruction be threatening life, tracheotomy must be done.

Chronic Laryngitis.

Causes.—It is often a sequel to repeated attacks of acute laryngitis, but it may be chronic from the first. The constant breathing of a dusty atmosphere,

as in the case of miners, and the constant over-use of the voice, as in the case of clergymen, actors, lecturers, and costermongers, are common causes. Alcoholism is another cause.

It is usual to class tubercle, syphilis, and cancer of the larynx with chronic laryngitis.

Symptoms and Physical Signs.—These are like those of acute laryngitis, but not so severe, although more persistent. There is constant hoarseness, with a hoarse cough, and pain on speaking and on swallowing. The sputum is muco-purulent.

In simple cases, improvement in the symptoms occurs under treatment, but recurrences are frequent. Laryngeal obstruction does not occur.

With the laryngoscope, the mucous membrane is seen to be swollen, granular, and covered by secretion.

In tubercular cases (sometimes called laryngeal phthisis), improvement for a time *may* occur if the patient be placed under favourable circumstances. But in most cases pulmonary phthisis is also present. The symptoms are the same as those of simple chronic laryngitis. Laryngeal obstruction is not common, because the tubercular nodules soften and break down before they are large enough to obstruct the passage. Perichondritis, with necrosis of the laryngeal cartilages, and expectoration of small pieces of cartilage, is fairly common.

With the laryngoscope, in the early stage, little pale nodules are seen in the mucous membrane, over the epiglottis, or at the back part of the ary-epiglottidean folds. These increase in number, forming little nodular heaps, which soon break down, and leave ulcers. Nearly the whole of the mucous membrane may eventually ulcerate.

In syphilitic cases, the symptoms are the same as those of the chronic form, but may come on more acutely. Laryngeal obstruction is not uncommon from sudden inflammatory œdema, and, in neglected cases, perichondritis, with necrosis of cartilage, may occur.

With the laryngoscope, in secondary syphilis, mucous tubercles, white patches, and superficial ulcers, may be seen; and in the tertiary stage serpiginous ulcers, with thickened edges, may be found on the ary-epiglottidean folds. Gummata are rare.

Cancer of the larynx is usually found on one or other *true cord*, and is, therefore, *squamous epithelioma*. The symptoms of ordinary chronic laryngitis come on slowly. Laryngeal obstruction usually occurs before the end, because the growth is at the rima glottidis (which is the narrowest part of the larynx), and gets large enough to block it. Perichondritis is also common. With the laryngoscope, in the early stage, a projecting nodule is seen on one of the true cords, and the movements of that cord become much impaired. As the nodule gets larger, its top ulcerates, and eventually the growth spreads all over the larynx, while the greater part of its surface becomes an ulcer.

Treatment.—In simple cases, try counter-irritation with iodine or blisters, externally; and internally apply astringents, such as glycerine of tannin, etc., on a brush. Medicinally the same drugs may be used as in acute cases, and the irritation may be allayed by linctuses or lozenges containing opium, cocaine, etc. Spraying the larynx with astringent, antiseptic or sedative solutions may be tried, or powders of a similar nature may be blown on to

the mucous membrane. Simple ulcers may be touched with nitrate of silver or sulphate of copper.

In tubercular cases, the general health must be attended to. Ulcers may be touched with nitrate of silver or solutions of lactic acid.

In syphilis, mercury and iodide of potassium must be given, and the same local applications may be tried as above.

In cancer, the growth, or, at a later stage, the whole larynx, has been removed with success.

Smoking, alcohol, and much speaking, are injurious in all laryngeal affections.

TUMOURS OF THE LARYNX.

The larynx may be the seat of innocent tumours as well as of cancer. The commonest of these is the papilloma, which occurs as a warty growth on the true cords, but does not impede the movements of the cord in the way that cancer does. Still, it is often necessary to remove a piece of the growth and place it under the microscope before a positive diagnosis can be made. Fibromata, myxomata, and adenomata, may also occur in the larynx, and are often polypoid. All growths in the larynx give rise to the symptoms of chronic laryngitis.

LARYNGISMUS STRIDULUS.

This is essentially a disease of young children. The Causes are unknown, but it is frequently associated with rickets and with congenital syphilis. In many cases the thinned spots in the cranial bones, called *craniotabes*, are present. These spots are most often found in the fossæ of the occipital

bone, and they are now considered to be evidences of congenital syphilis. It has been suggested that the spasm of the laryngeal muscles may be caused by pressure on the vagus or recurrent laryngeal nerve by enlarged lymphatic glands or a large thymus or thyroid, but this view is not established. Another view is that the spasm is excited reflexly by irritation of the gums during teething or of the intestine by worms.

Symptoms.—The disease is characterized by an occasional loud whooping or crowing sound, made by the child during inspiration. It is not preceded by the running cough of whooping-cough—in fact, there is no cough at all—and the other symptoms of laryngitis, such as hoarseness, are absent. The whooping sounds may be repeated frequently (in which case dyspnœa and cyanosis may be present) or at intervals. They are more common at night. Between the paroxysms the child is perfectly well.

The crowing sound is produced by inspiration through the glottis, which is almost completely closed by spasm of the adductor muscles of the larynx. In some cases spasmodic contractions of the hands and feet (carpo-pedal spasms) also occur, and sometimes even general convulsions.

The disease usually ends in recovery, but the spasms may recur at various periods of infantile life. In rare cases death has occurred suddenly from complete spasmodic closure of the glottis.

Treatment.—Treat the general health with ol. morrh. or syr. phosph. co., etc. The gums must also be attended to. If the spasms be frequent, or affect other parts besides the larynx, a hot bath should be given, and antispasmodics, such as belladonna, bromides, or chloral, may be prescribed.

PARALYSIS OF THE LARYNX.

This may occur in bulbar paralysis, in diphtheritic paralysis, or from the pressure of an aneurism on the recurrent laryngeal nerve (see p. 99).

Paralysis of the larynx is indicated by aphonia and the immobility of the vocal cords, as seen with the laryngoscope. As the patient cannot cough properly, particles of food may pass the larynx and set up a pneumonia which ends in gangrene.

THE RELATION OF CROUP TO DIPHTHERIA.

The word *croup* has been used very loosely to indicate any disease (except whooping-cough) in which a whooping noise is made in the throat. It has, consequently, been applied to simple laryngitis, membranous laryngitis, and laryngismus stridulus.

Now, there is an old controversy as to whether membranous croup, *i.e.*, membranous laryngitis, is the same disease as diphtheritic laryngitis or not. Those who think that the diseases are different assert that in membranous croup the fibrinous membrane is loosely attached, because it is formed on the surface of the mucous membrane by coagulation of the inflammatory exudation, while in diphtheria the membrane is closely adherent, because it is formed in the substance of the mucous membrane by the coagulation, not only of the exudation, but also of the epithelial cells of the mucous membrane. But this distinction will not hold, because, however adherent a diphtheritic membrane on the fauces may be, when it spreads

to the larynx or trachea it may be quite loosely attached.

Again, it has been asserted that membranous croup is never accompanied by albuminuria nor followed by paralysis; but, after all, the same may be said of many *mild* cases of diphtheritic laryngitis.

Consequently, although we cannot assert that there is no other cause except the diphtheritic poison which can produce a membranous laryngitis, still, it is believed that the vast majority of such cases are diphtheritic.

The discovery of the bacillus of diphtheria will settle this question, for if a number of cases of membranous laryngitis in which this bacillus is absent can be collected, the distinction between membranous croup and diphtheria will be established.

DISEASES OF THE LUNGS.

INFLAMMATION OF THE BRONCHIAL TUBES —BRONCHITIS.

Acute Bronchitis.

Primary Varieties: (1) Catarrhal; (2) Suppurative; (3) Fibrinous.

1. *Catarrhal Bronchitis.*

Causes.—This is the commonest variety of bronchitis, and it most frequently occurs in children and old people. It may be due to *cold*; or to the *specific fevers*, especially *measles* and *whooping-cough*; or to prolonged *congestion*, as in mitral disease; or to the inhalation of moderately irritating substances, such as pollen, volatile chemical irritants, etc.

Pathological Anatomy.—There are two stages: (1) The *dry* stage. In this the mucous membrane is hot, red, swollen, and dry. Microscopically the vessels are gorged with blood, the subepithelial connective tissue is crowded with exuded leucocytes, and the epithelial cells are swollen. (2) The *moist* stage. In this the epithelial cells burst and discharge mucus, the glands secrete abundant mucus, and the leucocytes work their way to the surface, which becomes covered by a muco-purulent secretion.

Pathological Terminations. — (1) Recovery, in which the secretion gradually stops. (2) Extension

to (a) the small bronchi, constituting *capillary bronchitis*, or even to (b) the air-vesicles, constituting *catarrhal lobular pneumonia*. In this case the air-vesicles become filled with epithelioid cells and leucocytes, so that certain lobules of the lung become *solid* (see p. 38). With capillary bronchitis and lobular pneumonia there are usually associated *compensatory emphysema* (see p. 33) and *lobular collapse* (see p. 45). (3) Chronic bronchitis.

Symptoms and Physical Signs.—There is a feeling of constriction across the chest, and a *pain behind the sternum*, at a point corresponding to the bifurcation of the trachea. The *pyrexia* and pyrexial symptoms, thirst, quick pulse, etc., are moderate. *Cough* is frequent and painful. The first characteristic, *sputum*, consists of tough pellets of thick muco-pus; later, when the moist stage sets in, the sputum becomes thin muco-pus and frothy from admixture with air-bubbles. The *dyspnœa* is moderate. In the dry stage the physical signs are dry râles, *i.e.*, rhonchi produced in the big tubes and sibilli in the smaller tubes. In the moist stage, if the secretion be abundant, moist râles, *i.e.*, crepitation, may be heard. These physical signs are audible over all parts of both lungs, but the moist sounds are most common at the bases, because the fluid tends to accumulate in the dependent parts. In a typical case resonance on percussion and vocal resonance are normal.

If the inflammation reaches the smallest tubes (capillary bronchitis), the crepitation becomes very fine, and there is more dyspnœa, and perhaps cyanosis. If lobular pneumonia occur, the crepitation becomes consonating, because it is heard

through the solid lobules (see p. 15), and the signs of asphyxia are still more marked. If the disease tends to become chronic, the cough and expectoration persist, but urgent symptoms do not arise.

Treatment.—Keep the patient in bed, put on a poultice over the sternum or over the whole chest, and let the patient inhale from a steam-kettle. Prescribe vin. ipecac. or vin. antimoniale with ammon. carb., tinct. camph. co., and infus. seneg. The above measures promote secretion, and consequently relieve the pain and the pain on coughing. When the moist stage sets in, if the secretion be moderate, it may be stopped by a little tinct. opii and acid. sulph. dil., and linctuses containing squills and balsam of tolu. But *if the secretion be abundant*, so as to cause much dyspnœa, or if capillary bronchitis supervenes, opium must on no account be given (see p. 4), but stimulant expectorants like spir. æth. sulph. and spir. chloroform., and cardiac tonics like digitalis and strychnia, must be chiefly relied on. Brandy must be given, and the poultices (which are now too heavy) must be replaced by turpentine stupes. The inhalation of steam must be stopped unless the secretion is still very thick. If there be cyanosis, oxygen may be inhaled, and, in young people, emetics may be prescribed and venesection employed. If the case tends to be chronic, dilute acids, opiates, copaiba, terebene, etc., may be used to stop the secretion. Dry inhalations of creasote, eucalyptol, etc., are also useful, and iodine may be painted over the chest. The patient should be encouraged to take deep breaths, so as to get the inspired air behind the secretion, and so dislodge it.

2. *Suppurative Bronchitis.*

This occurs when septic material (especially decomposing food) reaches the bronchi, in consequence of laryngeal paralysis, perforation of the trachea, etc. The mucous membrane is bright red and granular, and there is a copious secretion of pus. The inflammation spreads to the capillary tubes and air-vesicles, and so sets up a pneumonia, which soon ends in abscess or gangrene.

The **Symptoms** are those of bronchitis and pneumonia, with high fever and purulent sputum, and death is the usual result.

3. *Fibrinous Bronchitis—Plastic Bronchitis.*

This occurs in diphtheria and in a disease called plastic bronchitis. It is characterized by the death of the epithelium, and by an exudation which coagulates and deposits a solid layer of fibrin on the mucous membrane, thus forming a membranous cast of the bronchial tubes.

It is said that plastic bronchitis can be distinguished from diphtheritic bronchitis by the absence of the diphtheritic bacillus, and of albuminuria, paralysis, and the other toxic symptoms of diphtheria.

The **Symptoms** of plastic bronchitis are those of the catarrhal form, with great obstruction to the entrance of air into the lungs, and the expectoration of casts of the bronchi. There may be considerable hæmoptysis.

In addition to the ordinary remedies, iodide of potash may be given.

Chronic Bronchitis.

Causes.—It is most often a sequel to repeated attacks of acute catarrhal bronchitis; but it may be a chronic process from the first, and be due to the continued inhalation of mildly irritating particles, such as coal-dust (when it is called *anthracosis*), steel-filings (*siderosis*), etc.

Pathological Anatomy.—There is a slow, continuous proliferation of the epithelium of the bronchial tubes, with hyper-secretion of the mucous glands and a slow, continuous exudation of leucocytes. Consequently there is a constant supply of muco-purulent material (called *phlegm*) in the tubes. But, in addition, there is a growth of granulation tissue in the subepithelial connective tissue, and this granulation tissue develops into fibrous tissue, which makes the walls of the tubes thicker.

The granulation tissue may extend through the bronchial wall to the connective tissue outside it, when the condition is called *peribronchitis*; and, further still, it may extend into the connective tissue between the air-vesicles, in which case the condition is called *interstitial pneumonia* (p. 43). In both these conditions the granulation tissue develops to fibrous tissue, and the lung becomes very fibrous. In anthracosis the lung is black.

Complications.—Besides interstitial pneumonia, chronic bronchitis may lead to bronchiectasis (p. 31), and to emphysema (p. 33). Asthma is both a cause and a consequence of chronic bronchitis. In all cases the right ventricle becomes hypertrophied, and later on dilated.

Symptoms and Physical Signs.—There is a constant cough, with the expectoration of thin and

frothy or stringy muco-pus. If the sputum be very abundant and watery, the condition is called *bronchorrhœa*. The physical signs consist of rhonchi and sibilli, and, if the secretion be thin enough, crepitation. There is no dulness. There may be some fever. If the dyspnœa become urgent, it usually means that an acute attack has supervened on the chronic. The physical signs of emphysema (p. 34) and of bronchiectasis (p. 32) are often present.

Treatment.—Counter-irritation to the chest by iodine paint, etc., or an occasional poultice, may be tried. Internally give dilute acids, opium (if there is no dyspnœa), or copaiba. Squill, tolu, syrup of poppies, etc., are often soothing. Inhalations of tinct. benzoin. co., creasote, eucalyptol, terebene, etc., are very useful, and may be ordered with or without steam, according as the sputum is thick or thin. A warm, dry climate is usually the best.

Dilatation of the Bronchi—Bronchiectasis.

Causes.—1. **Bronchitis.**—Bronchitis leads to bronchiectasis (*a*) by weakening the resisting power of the bronchial walls; (*b*) by narrowing the lumen of the tubes (when the new submucous fibrous tissue contracts), and so damming up the secretion above; (*c*) by the dilating action of the constant cough; (*d*) by the supervention of interstitial pneumonia, because the new fibrous tissue then formed in the lungs contracts and drags open the adjacent tube.

2. **Pleurisy.**—In chronic pleurisy the fibrous tissue which forms the adhesions (p. 70) may extend into

the lung, and by contracting drag open a bronchial tube.

3. **Phthisis.**—When tubercles in the wall of a bronchial tube break down, the tube is, of course, dilated from ulceration. But in chronic phthisis there may be much formation of fibrous tissue, and bronchiectasis may result from its contraction.

Bronchiectases may be cylindrical, sacculated (when due to traction), or, in tuberculosis, ulcerated. With the exception of the last, bronchiectatic cavities can be distinguished from tubercular, abscess, or gangrenous cavities, by possessing a smooth lining, and by their definite communication with a bronchus.

Symptoms and Physical Signs.—These are those of chronic bronchitis, but the *sputum* is usually offensive; and it is common for a large quantity to be expectorated intermittently, say once a day. This is due to the fact that the dilated bronchus is insensitive, and tolerates the presence of sputum, until the quantity is large enough to stretch the wall. This excites a cough which expels all its contents.

If the dilated tube be large and near the chest-wall, it may give rise to a boxy note on percussion, and to amphoric breathing and vocal resonance when it is empty. But when it is full, there will be dulness, and loss of breath-sounds and of vocal resonance over it. Such a change in the physical signs is very characteristic of bronchiectasis.

Treatment.—The same as that of chronic bronchitis; but it is especially necessary to use anti-septic inhalations. If near the surface, an attempt may be made to open the cavity through the chest-wall, and drain it.

Emphysema.

The presence of air-bubbles in the connective tissue of the lungs is called *interstitial* emphysema. It may follow the rupture of the air-vesicles in whooping-cough or the laceration of the lung by a broken rib, etc. But the common emphysema is **vesicular emphysema**, and consists in the over-distension of the *air-vesicles* with air.

Vesicular emphysema may be produced in two ways :

1. A small part of the lung becomes incapable of expanding on account of lobular pneumonia or collapse, etc. Then, on full *inspiration*, the adjacent normal air-vesicles become stretched by the entering air, so as to fill up the space which the non-expanding air-vesicles should have occupied. Otherwise a vacuum would occur between the solid lobule and the chest-wall.

Emphysema produced in this way is called *inspiratory* emphysema, because the stretching of the air-vesicles occurs during inspiration. It is also called *compensatory* emphysema, because the over-distension of certain air-vesicles compensates in area for the non-expansion of others. In distribution it is always patchy, being situated around the solid areas (p. 38).

2. When a forcible expiration is made through the narrowed bronchial tubes, as in chronic bronchitis and asthma, or through a narrow orifice, as in playing wind-instruments or in glass-blowing, the air in the air-vesicles is compressed. Now, those air-vesicles which are supported by the ribs do not yield to this pressure, but those at the apex of the lung and at the anterior border (where the

costal cartilages lie) are not so supported, and consequently they *give*, and become over-distended. This is called *expiratory* emphysema.

In both inspiratory and expiratory emphysema the walls of the over-distended air-vesicles soon atrophy, and *microscopically* they are seen to be very thin, and to have lost their elastic fibres and capillaries. They may even be quite worn away, so that one air-vesicle communicates with another.

Macroscopically the emphysematous parts of the lung bulge from over-distension, and overlap the heart considerably. They do not collapse, because their elasticity is lost, and they are pale from the absence of bloodvessels. The dilated air-spaces are mapped out on the surface of the lung, and it is easy to press the air from one part of the emphysematous portion to another. The right ventricle is hypertrophied and dilated.

Most emphysematous lungs are large, and have, therefore, been improperly called *hypertrophous*. Occasionally, especially in old people, the lung is small, and this has been called *atrophous* emphysema.

Symptoms and Physical Signs.—There is habitual dyspnœa, aggravated by exertion. A loud expiratory wheeze on going upstairs is very characteristic. There are dilated veins on the cheeks, and sometimes the whole face is purple.

On *inspection* the chest is usually *barrel-shaped*, from lengthening of the antero-posterior diameter; but sometimes it is very long and flat, with projecting scapulæ. The expansion is always bad, so that during inspiration the chest, as a whole, is lifted up, but does not expand laterally. The extraordinary muscles of respiration may be seen to

be hypertrophied. Epigastric pulsation is often visible on account of the dilatation of the right ventricle. On *percussion* the lung is hyper-resonant, and, as it overlaps the heart, cardiac dulness is diminished or lost. Hepatic dulness also is diminished.

On *auscultation*, expiration is *prolonged*, because the lung has lost its elasticity. The second sound over the pulmonary valves is accentuated, because the tension in the pulmonary artery is high, on account of the diminution in the number of capillaries in the lungs.

If bronchitis be present, there is cough with expectoration, and râles will be heard on auscultation.

Treatment.—As the lungs contain an excess of residual air, breathing into rarefied air has been recommended. As a matter of fact, emphysematous people usually breathe most easily at high altitudes. For the rest, the treatment must be that of chronic bronchitis. Cod-liver-oil and the syrup of the phosphates are useful.

ASTHMA.

In asthma the bronchial tubes become narrowed, so that great dyspnœa results.

Pathology.—Some authorities think that the narrowing of the tubes is due to spasm of the muscle in the wall of the bronchi. According to this view, certain persons are born with, or acquire (through repeated attacks of bronchitis), an irritable condition of the bronchial muscular fibres, which makes them prone to spasmodic contraction. Given this irritability, the actual spasm may be excited

(a) *directly*, by the inhalation of pollen (as in hay asthma), dust, cold or damp air, etc. ; (b) *reflexly*, by irritation of the stomach (dyspepsia) or fauces (post-nasal adenoids), ovaries, etc. ; (c) *centrally*, by mental emotions, or poisonous blood circulating in the medulla, as in uræmia.

In favour of this view are the facts that the onset of the attack is very sudden, that the disease is most common in neurotic persons, and that antispasmodic remedies sometimes stop the attack instantaneously.

The other view is that the narrowing is due to a sudden congestion of the bronchial mucous membrane. This is supported by the fact that there is a characteristic secretion.

Symptoms and Physical Signs.—The first attack may occur in childhood, or at any later period. Most often it comes on at about two o'clock in the morning. There is great difficulty in breathing, but the respirations are too laborious to be very frequent. The feeling of want of breath is very acute, and the patient uses all the voluntary muscles of respiration. Usually he leans forward and grasps the bedrail, or some firm support, so as to fix the shoulders. The face may be blue. In breathing, the *inspiration* is short, because ineffectual, the air being unable to enter the lungs. In young persons the flexible parts of the chest-wall, such as the costal cartilages and lower ribs, may even recede during inspiration. *Expiration*, on the other hand, is very prolonged, and gives rise to a loud wheezing sound. An attack may last a few hours, or, with partial relaxations, for days.

Asthma always leads to emphysema, and is associated with bronchitis, so that the symptoms and

physical signs of these affections are often present. In old asthmatics the face is permanently cyanosed, the shoulders become rounded, and the scaleni hypertrophied.

Prognosis.—Asthma is not fatal of itself, but may lead to death from bronchitis and emphysema. Children may cease to have attacks as they get older, but if the attacks become more frequent between twenty and thirty years of age, the prognosis is bad.

Treatment.—Attacks may be stopped by inhaling the smoke of stramonium cigarettes or nitre-papers. Internally, stramonium, lobelia, and sulphuric ether are often useful, and the habitual administration of iodide of potassium is recommended. As antispasmodics, nitrite of amyl and nitroglycerine are worth trying.

For chronic cases a suitable climate must be found, or, rather, a suitable atmosphere, for a change from one street to another of the same town may cause the attacks to cease. It is impossible to predict the kind of atmosphere which will suit a particular case. Some asthmatics can breathe easily in a fog, but cannot breathe on a cold, clear day, and *vice versa*. Hence we must be guided by the patient's past experience. As a general rule, a damp, stagnant atmosphere is the worst.

INFLAMMATION OF THE LUNG SUBSTANCE —PNEUMONIA.

Pneumonia is divided, according to the distribution of the pneumonic areas in the lung, into lobular and lobar.

Lobular Pneumonia.

This is also called broncho-pneumonia, because it is nearly always an extension of bronchitis (p. 27). It is also often called catarrhal pneumonia, because the inflammation (like that of the bronchi) is most often a catarrhal one, *i.e.*, there is a proliferation of epithelium, and not a formation of fibrin or pus. Catarrhal pneumonia is most common in children.

The Causes are the same as those of bronchitis.

Pathological Anatomy.—*Macroscopically* the lung is studded with areas which are solid, pyramidal in shape, red, and swollen. These are the pneumonic lobules. Around these there are areas of compensatory emphysema (p. 33), the air-vesicles in which are distended with air, and pale. Capillary bronchitis being usually also present, the bronchioles are filled with muco-pus. Very often there are also areas of lobular collapse in these lungs (p. 45).

Microscopically, in catarrhal lobular pneumonia, the air-vesicles are filled with epithelioid cells (detached from the walls) and exuded leucocytes.

Symptoms and Physical Signs.—As a rule, the symptoms and physical signs of bronchitis occur first. When the pneumonia supervenes, there may be less resonance on percussion, and the vocal resonance and fremitus may be increased because of the numerous solid areas in the lung. But the most characteristic physical sign is *consonating crepitation* (p. 15). At the same time there is great dyspnoea, and perhaps cyanosis. The temperature may not be much higher than in capillary bronchitis. If several adjacent lobules become

solidified, there will be dulness, tubular breathing, and bronchophony over them.

The **Treatment** is the same as that for grave cases of bronchitis (p. 28).

Lobar Pneumonia—Croupous Pneumonia.

Causes.—This is now considered a specific disease, due to the *Pneumococcus*.

The *Pneumococcus* is an oval-shaped bacterium which is often found in groups of two (diplo-cocci). It can be cultivated, and from the cultivations the poison produced by the bacterium can be obtained. This poison is called *pneumotoxin*, and, by cultivating the same organism in a special way, an antidote to this poison, called *antipneumotoxin*, can be isolated. It is maintained that, by injections of antipneumotoxin, the crisis of croupous pneumonia (which means the cure of the disease) can be brought about.

The *predisposing* causes of croupous pneumonia are cold and wet, alcoholism, debility, and the specific fevers.

Pathological Anatomy.—It must be understood that *croupous* in this connection means *fibrinous*. In fibrinous pneumonia a lobe, or a still greater part of the lung, is affected. The disease is divided into three stages :

1. **Engorgement.**—This stage has been called splenization, because the lung is thought to look like the spleen. The lung is redder and larger than normal, but still floats. On pressure, a thick red juice exudes. Microscopically the vessels in the walls of the air-vesicles are gorged with blood.

2. **Red Hepatization** (so called because the lung is thought to resemble the liver).—In this stage the exudation from the bloodvessels has coagulated to form fibrin. The affected lobe is solid (sinking in water), swollen, red, comparatively dry, and finely granular on section. It tears more easily than normal. The pleura over the affected lobe is covered with lymph from co-existing pleurisy. Microscopically the air-vesicles are filled with fibrin, red blood-discs, and leucocytes.

3. **Gray Hepatization**.—In favourable cases the fibrin becomes infiltrated by leucocytes, and so broken up as to be capable of being expectorated or absorbed. This is called *resolution*. In unfavourable cases the leucocytes and serum continue to be poured out, and thus infiltrate the lung with a purulent fluid. The lung is, then, still solid enough to sink in water, and it is swollen and dirty gray. It can scarcely be touched without tearing, and when squeezed the pus pours from it. This is the true gray hepatization, but it is best called *purulent infiltration*. It readily goes on to *abscess* or *gangrene*. Microscopically both the air-vesicles and their walls are filled with necrotic leucocytes.

In rare cases, granulation tissue grows in the walls of the air-vesicles, and then the condition becomes one of chronic interstitial pneumonia.

Symptoms.—The disease begins acutely with a severe rigor, vomiting, and a rapid rise of temperature to 104° or more. There is urgent dyspnoea, so that the pulse-respiration ratio is not maintained. A sharp pain (due to the pleurisy) is felt on the affected side, and there is a cough. The face is flushed, and the pulse full and bounding, croupous pneumonia being the very type of a sthenic disease.

The urine is febrile, and the skin is exceptionally hot and dry.

The cough is for a short time dry, but soon the typical sputum is brought up. It consists of tough, tenacious, *rusty-coloured* pellets, composed of fibrin, red blood-discs, and leucocytes. In bad cases the sputum becomes more fluid and more bloody, and of the colour of plum-juice.

Herpes on the lips or cheek is a common complication.

The temperature keeps high without remissions all through the disease, but the dyspnœa becomes less urgent after about twenty-four hours. The chlorides may be completely absent from the urine. In favourable cases *crisis* occurs in about five days, *i.e.*, the temperature falls to normal suddenly, there is profuse sweating, and all the symptoms improve, although the physical signs take a long time to clear up.

In unfavourable cases the fever becomes remittent, the dyspnœa more urgent again, and delirium may set in, especially in alcoholic persons. The pulse may remain full and strong to within an hour of death. If gangrene occurs, the breath becomes fœtid.

Physical Signs.—In the stage of engorgement the only physical sign is *very fine crepitation*, perhaps only heard on taking a deep inspiration (p. 14).

In red hepatization there is *dulness*, *tubular breathing*, *bronchophony*, and *increased vocal resonance* over the affected lobe.

During resolution the dulness, bronchophony, and increased vocal resonance gradually disappear, while the tubular breathing becomes mixed with, and then replaced by, *consonating crepitation*. This

is called *redux* crepitation, and it is gradually replaced by vesicular, *i.e.*, normal, breathing.

If purulent infiltration occur, the dulness persists, and the tubular breathing becomes replaced by consonating crepitation, which spreads all over the lung instead of clearing up.

Prognosis.—This is, generally speaking, good, but is bad in old and in alcoholic persons. A high temperature, albuminuria, and the plum-juicesputum are evil signs, and apical pneumonia (in which the upper lobe is affected) is more fatal than basic.

Treatment.—In strong young persons the urgent dyspnoea and threatened asphyxia may be treated by venesection. In other cases it is better to give a simple diaphoretic, with vin. ipecac. and liq. ammon. acetat., and put on a jacket poultice. If unfavourable symptoms arise, brandy, ether, ammonia, strychnia, and digitalis must be given, and the poultices must be replaced by stupes. Hyperpyrexia may be treated with quinine or with ice-packs.

Varieties of Croupous Pneumonia.—*Abortive or Larval Pneumonia* means that the crisis occurs early, and the physical signs are slight. *Cerebral pneumonia* means that delirium, squinting, and other cerebral symptoms, are prominent, perhaps so much so as to cause the lung disease to be overlooked (*e.g.*, in children meningitis may be diagnosed, and in drunkards delirium tremens). *Massive pneumonia* means that the tubes as well as the air-vesicles are filled with fibrin. In such cases there is no tubular breathing (p. 13), and the case simulates pleuritic effusion, except that the sputum is rusty and the temperature very high.

Chronic Interstitial Pneumonia.

This disease consists in the growth of granulation tissue in the walls of the air-vesicles. The granulation tissue becomes fibrous tissue, so that the lungs become hard and traversed by white bands, which in anthracosis may be blackened by particles of coal-dust. Some parts of the lung remain emphysematous, and bronchiectases (p. 31) are often present.

This growth of granulation tissue most often begins round the bronchi in chronic bronchitis, especially when due to anthracosis, siderosis, etc. (p. 30), and from thence it extends to the air-vesicles. But it may spread in from the pleura in chronic pleurisy (p. 55), and may even *begin* in the walls of the air-vesicles in cases of croupous pneumonia in which resolution is delayed. In cases of congenital syphilis, a considerable part of the lung may be found solid and white, and, microscopically, this is seen to be due to interstitial pneumonia.

The **Symptoms and Physical Signs** will be those of the conditions which give rise to the interstitial change, *i.e.*, chronic bronchitis, pleurisy, and pneumonia. Very often there is evidence of bronchiectasis (p. 31).

This disease has been called fibroid phthisis, because the fever, emaciation, etc., cause it to resemble phthisis. The signs of a cavity, too, may be present, only in this case it is a bronchiectasis. The absence of tubercular bacilli will, however, settle the diagnosis, and it seems advisable to restrict the term phthisis to tubercular cases (p. 47). The disease has also been called cirrhosis of the lung.

ABSCESS OF THE LUNGS.

Pathology.—Abscesses in the lungs result from very severe croupous pneumonia (p. 39); from the lobular pneumonia which follows suppurative bronchitis; and from the suppuration of infarcts (p. 46) when the embolus comes from a septic source, as in pyæmic thrombosis. Pyæmia may also cause pulmonary abscesses without there being a definite infarct. An abscess cavity has soft, irregular walls.

Symptoms and Physical Signs.—These are those of pneumonia, either lobar or lobular (see pp. 38, 39). But the fever is remittent, and there are rigors and sweatings, and the sputum is purulent. There may be the physical signs of a cavity.

The **Treatment** is that of pneumonia. It might be possible to open the abscess through the chest-wall.

GANGRENE OF THE LUNG.

This means the death and putrefaction of a piece of lung tissue, and it usually is produced by a pneumonia of great severity, especially the pneumonia set up by the entrance of decomposing material into the air-passages. Infarctions also become gangrenous if the embolus has come from a putrefactive centre. In gangrene, a soft, blackened, offensive slough, composed chiefly of dead blood-vessels, is found in the lung. It lies in a cavity filled with gay pus and bounded by soft, irregular walls, composed of pneumonic lung.

The **Symptoms and Physical Signs** are the same as those of abscess of the lung, but in addition the odour of the breath is characteristically offensive.

ATELECTASIS—COLLAPSE OF THE LUNG.

Atelectasis is the name given to unexpanded fœtal lung. It may be partial or complete. The affected portion is solid, small, and dark-coloured, while the expanded areas are pink.

Acquired Collapse may be lobular or lobar.

Lobular Collapse results from capillary bronchitis (p. 27), because if the secretion completely blocks a tube, the air in the lobule behind the block becomes absorbed until the air-vesicles are empty. Another view is that the secretion in the tube acts like a ball-valve, and allows the air to pass during expiration, but prevents its entry during inspiration, until the air in the lobule is exhausted. In lobular collapse the affected lobule is pyramidal in shape, solid, slate-coloured, and shrunken. The last two attributes distinguish it from lobular pneumonia, especially on section.

Lobar Collapse is due to the compression of the lung by fluid in the pleura or pericardium, or by solid tumours and aneurisms. The lung is solid, shrunken, and dark-coloured. On section it is very smooth.

Symptoms and Physical Signs.—These are masked by those of the affections which cause the collapse. Thus, in lobular collapse the symptoms and physical signs of capillary bronchitis are present; and in lobar collapse those of pleuritic effusion, etc. But if a large piece of collapsed lung is in contact with the chest-wall, and the tubes in it are open, dulness, tubular breathing, bronchophony, and increased vocal fremitus, will be obtained over it. This sometimes occurs in large pericardial effusions.

PULMONARY APOPLEXY — HÆMORRHAGIC INFARCTION—PULMONARY EMBOLISM.

Pulmonary apoplexy means hæmorrhage into the lung tissue, as distinguished from hæmorrhage from a cavity. It is nearly always the result of hæmorrhagic infarction, but diffuse hæmorrhages may occur in scurvy, malignant fevers, etc.

A hæmorrhagic infarction in the lungs is produced as follows: A terminal artery becomes blocked by an embolus, and the piece of tissue supplied by it anæmic. Then the blood regurgitates from the veins of the surrounding pieces of lung into the empty capillaries of the affected piece. But these capillaries have, while empty, become so damaged as to be unable to hold the blood, so that they rupture, and the blood which they contain is poured out into the adjacent air-vesicles. An infarction is pyramidal in shape, swollen, and solid. It therefore resembles a patch of lobular pneumonia (p. 38), but it is of a deep black-purple colour, like that of damson cheese. Microscopically its air-vesicles are simply full of blood.

The emboli which block the arteries are most often pieces of fibrin detached from a thrombus in a vein (as so often occurs in the puerperal state), or in the right side of the heart. If the thrombus be septic, the area of the infarction suppurates. Gaseous emboli also occur in the lungs from the accidental entrance of air into a vein, and fatty emboli (oil-globules) have been found after fractures of long bones.

Symptoms.—The impaction of an embolus in a branch of the pulmonary artery is indicated by a sudden pain in the side, and perhaps a rigor and

sudden rise of temperature. As the infarction forms there is often hæmoptysis.

Physical Signs.—If the infarction be large, dulness, tubular breathing, and bronchophony, may be found over it, because it is solid. If it be small, there may be no physical signs except a little consonating crepitation due to the presence of secretion in the neighbouring tubes.

If the main pulmonary artery be blocked by an embolus, there is sudden fearful dyspnœa, while all the time the air is entering the lungs freely. The face may be cyanosed or pale, and the pulse almost imperceptible. A systolic bruit may be heard over the pulmonary artery, if the block be not complete. Death often occurs in a few minutes.

PHTHISIS—TUBERCULAR PNEUMONIA.

Phthisis is an old clinical name for a *wasting* disease, but it is now settled that all cases of pulmonary phthisis are due to a *local tuberculosis*, accompanied by more or less *pneumonia*. The term fibroid phthisis is explained elsewhere (p. 43).

Causes.—The *immediate* cause of phthisis is the *tubercular bacillus*. The *predisposing* causes are *heredity*; *debility*, after a long fever, like typhoid, or from alcoholism, privation, etc.; residence in damp, badly-drained localities; and the presence of some old lesion of the lung, such as a patch of broncho-pneumonia, etc.

What is meant by the *tubercular diathesis* is a condition of the tissues which favours the growth of tubercular bacilli.

Pathological Anatomy.—There are various stages.

1. **The Stage of Discrete Tuberculosis.**—Bacilli are deposited in the infundibula of the air-vesicles (usually at the apex of the lung), and, in consequence, tubercles develop in that position. Each tubercle consists of a giant-cell, surrounded by epithelioid cells, and outside these is a zone of small round cells.

2. **The Stage of Consolidation and Caseation.**—The air-vesicles adjacent to the tubercles become filled with epithelium (catarrhal pneumonia), and the vesicular walls become infiltrated with leucocytes (interstitial pneumonia). Hence solid areas, consisting of tubercles and pneumonia, are formed, and the tubercles (from absence of bloodvessels) and the loose epithelium in the air-vesicles soon die and become caseous, and therefore yellow.

This stage is very transient, because, after caseation, one of the three following processes rapidly ensues :

(a) In rare cases granulation tissue develops round the caseous material, encapsules it with fibrous tissue, and stops the further progress of the disease. This is called the *healing* of tubercle.

(b) In some cases rapid suppuration occurs round the caseous material which, being exposed to moisture, becomes soft. The vesicular walls die, and are coughed up with the soft caseous material. In this way a **cavity** is formed. At the same time the tubercles spread all over the lungs, and rapidly break down. This is popularly called *galloping consumption*.

(c) In most cases the two processes just described alternate, so that at one time, under favourable circumstances, fibrous tissue is formed in the endeavour to stop the tuberculosis; while at other

times, under unfavourable circumstances, breaking-down occurs. Such cases last about two or three years, but sometimes they progress so slowly that they may last twenty years, by which time the amount of fibrous tissue in the lungs is very great, and the case may be considered a true fibroid phthisis. The processes described under (b) and (c) constitute the third stage of phthisis, which is that of softening to form cavities.

Post-mortem, a phthisical cavity is irregular in shape, and may be crossed by cords which are bloodvessels which have not yet softened. Sometimes there are small aneurisms on these vessels. From the wall of the cavity project the remains of bloodvessels, and all around it the lung is a mass of yellow and gray areas in various stages of softening. Further off still the lung is studded with discrete tubercles.

Pleuritic adhesions always form in phthisis. Sometimes, in acute cases, the softened areas burst into the pleura, causing pneumo-thorax.

As regards the rest of the body, tubercles may be found in any or all of the organs, most frequently in the larynx or intestine. The liver may be fatty or lardaceous. The spleen, kidneys, and intestines may be lardaceous.

Symptoms and Physical Signs.—In the first stage the patient feels ill, and there is slight *fever* of a *remittent* type, with some *sweating* as the temperature is falling. He has no sense of dyspnœa, but the rate of breathing may be quickened. There is a *cough*, but the *sputum* is scanty. Still, it may contain *bacilli* and *blood*. Some cases begin with a profuse *hæmoptysis*, and these run a very rapid course.

The physical signs are very vague, but a *jerkiness* in the inspiratory sound and *slight crepitation* at a localized spot may indicate the deposit of the tubercle.

The second stage may be unrecognisable as a distinct stage because the solid areas in the lung break down before they become large, and so the disease reaches the third stage. But if there be a large solid mass, the physical signs over it will be *dulness*, *tubular breathing*, *bronchophony*, and *increased vocal fremitus*. The general symptoms become somewhat aggravated.

In the **third** stage, that of softening, there is severe *remittent fever* with profuse *night-sweats*, and a *hectic flush* on the cheeks. *Emaciation* and *loss of strength* are obvious, and the *dyspnœa* may be appreciable. The *cough* is frequent, and the *sputum* is muco-purulent, and may be *nummulated* (p. 6), and it is often streaked with *blood*. It contains leucocytes, epithelial cells (from the air-vesicles), elastic fibres (from the vesicular walls), and *tubercular bacilli*.

The blood usually comes from the capillaries of the lung or of the granulation tissue round the tubercles; but sometimes an artery gives way, particularly if there be an aneurism on it (p. 49).

The physical signs in this stage are *dulness* with *consonating crepitation*. Sometimes a tubular whiff may be heard if a tube happens to be empty. There is also *bronchophony* and *increased vocal fremitus*, and the expansion of the affected side is diminished.

If a definite **cavity** has formed, the note on percussion becomes *boxy* or *cracked-pot* (p. 11). The breathing is *cavernous*, and the vocal resonance is

either *pectoriloquy* (if the cavity be small) or *cavernous*. The vocal fremitus is increased, and the expansion much diminished. In many cases there is a distinct depression in the chest-wall over the cavity.

Acute phthisis is characterized by the severity of all the symptoms, and particularly by the rapid extension of consonating crepitation all over the lungs.

In chronic phthisis the symptoms progress with occasional improvements, succeeded by exacerbations. The ends of the fingers become bulbous, and the nails curve sharply over them. In women there is amenorrhœa.

The end is often accelerated by diarrhœa (which is due to tubercular or lardaceous disease of the intestines), or by pneumo-thorax, or by a profuse hæmoptysis. Fistula in ano may occur.

Diagnosis.—In the early stages phthisis may be taken for broncho-pneumonia, but the latter is usually in both lungs, while phthisis is localized. In the later stages it might be taken for chronic interstitial pneumonia. In all cases the presence of tubercular bacilli in the sputum renders the diagnosis easy.

The **Prognosis** is very bad when there is a strong family history of tuberculosis, and in alcoholic persons and diabetics. Abundant crepitation, high fever, hæmoptysis, and diarrhœa are of evil significance. The only favourable cases are those which have no family history, and which can be placed permanently under favourable conditions.

The **Treatment** of phthisis may be radical or expectant.

1. The radical treatment consists in attempting to stop the disease by killing the bacilli. With

this object antiseptic drugs (such as mercury, creasote, guaiacol, etc.) and antiseptic inhalations may be ordered. Efforts are also being made to obtain from cultivations of the tubercular bacillus a substance which will act as an antitoxin, and kill the bacilli. It was hoped that Koch's tuberculin, although not an antitoxin, would destroy the tubercles by exciting inflammation around them, but this treatment has not succeeded.

2. The expectant treatment consists in strengthening the patient, and so enabling his granulation tissue to develop and cut off the tubercles. This is done by giving him cod-liver-oil, maltine, phosphates, etc., and sending him to a suitable climate. At the same time his symptoms must be treated.

The *fever* may be controlled by giving quinine, antipyrin, or phenacetin, shortly before the hour at which the temperature usually rises. External cold also is useful, and in some health-resorts arrangements are made for patients to sleep in the open air, and this is found to keep the temperature down.

Perhaps the best way to treat the *night-sweats* is to treat the temperature, but ext. belladonnæ, $\frac{1}{4}$ grain, in a pill is often useful.

For the treatment of *cough* and *hæmoptysis* see the rules laid down on pp. 6 and 8.

Pain in the chest may be treated by opium or by counter-irritation, and *diarrhœa* by opium, bismuth, and chalk.

Climate.—For consumptives the air must be *pure* (that is, free from micro-organisms and from dust) and *dry*. Rarefied air is beneficial, but temperature is not so important as long as it does not vary too much. Some authorities recommend a very cold atmosphere.

Sea-air is good because it is pure, but it is too moist for many cases, and a sea-voyage should never be ordered if there are numerous moist râles to be heard. Thickly populated districts are always to be avoided, because the air cannot be pure.

The disadvantage of many places where the air is dry is that they are dusty. This applies to many parts of South Africa, but there are other parts in which the advantages of this climate can be obtained without the inconvenience of the dust.

High altitudes are good because they are bracing, and the air is pure and rarefied; but a high table-land like Davos Platz in Switzerland and Colorado in North America must be selected, because a district in which mountain and valley alternate is apt to be rainy and swept by winds.

The climate of the Riviera, Egypt, and Algeria is suitable, but a crowded hotel must be avoided. Madeira and the Canary Islands are also good places.

In England we have to be content with Bournemouth and Ventnor, which are sheltered from the east wind, and dry quickly after the rain.

SYPHILIS OF THE LUNGS.

Syphilis may cause a chronic interstitial pneumonia with or without gummata. The latter usually appear as caseous nodules surrounded by fibrous tissue which has contracted and causes a depression on the surface of the lung. In congenital syphilis, chronic interstitial pneumonia may be found, the lung being white and solid (p. 43).

The **Symptoms** and **Physical Signs** are those of chronic interstitial pneumonia or of tumour; and mercury and iodide of potassium must be given.

TUMOURS OF THE LUNG.

These are usually cancerous or sarcomatous, and secondary to similar growths elsewhere. Sarcoma is the more common, because the infection of sarcomata travels by the bloodvessels, and reaches the lung early. They form whitish tumours, which are often the seat of hæmorrhage. Pieces of cartilage may be found in large tumours of the lung.

Symptoms and Physical Signs.—There is cough (with currant-jelly-like sputum), emaciation, and dyspnœa. There may be no fever. The diagnosis usually depends on the presence of a primary growth elsewhere, or the history of the removal of such a growth. If the growth in the lung be large enough, there will be dulness over it, and if open tubes traverse it, tubular breathing and bronchophony will be heard. If the tubes be occluded, the breath-sounds will be absent, and the case will simulate pleuritic effusion, and it is necessary to put in an exploring needle. A microscopical examination of the sputum might discover a portion of tumour, recognisable as such.

DISEASES OF THE PLEURA.

INFLAMMATION OF THE PLEURA—PLEURISY —EMPYEMA.

Causes. — Cold; certain fevers, particularly septicæmia and rheumatic fever; kidney disease; extension of inflammation from the lungs, liver, etc.; perforation from the lungs, œsophagus, etc.

Pathological Anatomy.—The bloodvessels become gorged, the endothelium dies and is shed, and leucocytes and plasma are exuded. The surface is now red, and has lost its polish. Then the plasma coagulates, depositing *fibrin* on the surface of the pleura, while serum accumulates in the cavity. From the pressure of the serum the lung becomes collapsed.

Terminations.—As a rule, the serum is absorbed, and granulation tissue develops beneath the fibrin and grows into it, and thus replaces it. Then the granulation tissues on the visceral and costal surfaces unite and become fibrous tissue, thus forming a fibrous adhesion across the pleura. Sometimes the granulation tissue extends into the lung, in the walls of the air-vesicles, producing interstitial pneumonia (p. 43). Suppuration sometimes occurs from the presence of pyogenic organisms. It is thought that complete resolution (*i.e.*, the removal of the inflammatory products and restoration of the endothelium without adhesion) may take place in a few cases.

Symptoms.—There is moderate *fever*, with all the febrile symptoms. A sharp *pain* in the side is felt, and it is aggravated by a deep breath, by coughing, or by pressure. *Dyspnœa* is present, because the pain makes the breathing superficial. The *cough* is dry and peculiar in character, because the patient tries to suppress it on account of the pain. When the fluid is exuded the pain is less, but the dyspnœa persists because the lung is compressed.

Physical Signs.—At first there is a *friction* sound heard both with inspiration and expiration. The patient avoids lying on the affected side on account of the pain. When the fluid has collected the signs are as follows :

Over the fluid (*i.e.*, at the base of the lung) there is dulness, loss of breath-sounds and of vocal resonance and fremitus.

At the level of the fluid ægophony is heard on speaking.

Above the fluid there is hyper-resonance (Skoda's note), and increased breath-sounds (puerile breathing), vocal resonance, and fremitus.

In addition there is diminished movement on the affected side, and increased measurement. The ribs are more widely separated, and the intercostal spaces may bulge. Lastly, the apex beat of the heart is displaced, being pushed away by the fluid, and on the right side the liver may be depressed. The patient now lies on the affected side, so as to give free play for the movements of the sound side.

As the fluid is absorbed the friction sound and the pain return, unless firm adhesions have formed.

If pus forms, the condition is called **Empyema**. The fever is then usually, but not always, higher, and very remittent, and there may be rigors and sweatings. The physical signs are practically the same as when the fluid is serum, but often, especially in children, some tubular breathing may be heard, instead of the complete loss of breath-sounds. In most cases an exploratory needle must be used to discover the nature of the fluid.

An empyema sometimes points and bursts through the chest-wall. Ordinary pleurisy usually ends in recovery in a few weeks, but sometimes it becomes chronic. In some of these cases the fluid is absorbed and dense masses of fibrous tissue form between the chest-wall and lung. The affected side then becomes contracted, and may measure

less than the sound side. The apex beat of the heart, too, is now drawn to the affected side, instead of pushed away from it, and the intercostal spaces are narrowed and retracted. The affected base remains dull, and the breathing over it feeble, but the vocal resonance and fremitus are not so diminished as when the fluid was present. Eventually even lateral curvature of the spine may result, from the contraction of the fibrous tissue.

Treatment.—At first give a simple diaphoretic mixture. If there be much pain, add to this liq. morph. acetat. At the same time poultices or leeches may be applied, or the affected side may be strapped. When the fluid is poured out, diuretics and watery purges are recommended in order to remove it vicariously, and iodide of potash may be used as an absorbent. Blisters are also recommended. If the fluid shows no diminution after a fortnight, draw it off with an aspirator, as, if it is left too long, the collapsed lung will not expand when the pressure is taken off.

An empyema must be opened and drained.

PNEUMO-THORAX.

This term means that there is air in the pleura.

Causes.—Rupture of a phthisical cavity, or of an abscess, or of a gangrenous cavity, into the pleura; perforating wounds of the chest-wall.

Symptoms.—There is a sudden pain in the side, accompanied by dyspnoea and the symptoms of shock.

Physical Signs.—In pure pneumo-thorax there is hyper-resonance all over the lung. If the hole which admitted the air become closed, the breath-sounds are absent; but if it remain open, the breath-sounds and the voice are amphoric.

Pyo-Pneumo-Thorax. — In most cases the air excites inflammation, and fluid (usually pus) accumulates at the bottom of the pleura. Then the signs of pneumo-thorax occur at the top, while below there are *all* the signs of fluid (p. 56). But the upper limit of dulness changes with the position of the patient, because the air always rises to the top.

In addition, a mixture of air and fluid in the pleura gives rise to a splashing sound on shaking the patient's shoulders. Metallic tinkling may, also, be heard on auscultation.

Treatment.—The pain and cardiac failure may be treated with opium and brandy. In some cases the pleura may be opened.

HYDRO-THORAX.

In this condition there is a passive effusion of serum into the pleura. It occurs in cardiac and renal dropsy and when the veins at the root of the lung are compressed.

Post mortem it can be distinguished from pleuritic effusion by the fact that the fluid is clear while the surface of the pleura is smooth and not covered by fibrin.

During life there are all the signs of fluid at the base of the pleura, but no friction, no cough, and no fever.

DISEASES OF THE PERICARDIUM, HEART, AND GREAT VESSELS.

GENERAL CONSIDERATIONS.

In all these cases the *physical signs* obtainable over the heart and great vessels are of prime importance, and the different phenomena known as *syncope*, *palpitation*, *tachycardia*, and *bradycardia* must be considered with these. An examination of the *pulse* is also essential. In every case, too, the presence or absence of *back pressure symptoms* must be noted, and of these *dyspnœa* and *dropsy* are the most important.

The Physical Signs of the Heart, etc.

1. *Inspection*.—A *bulging* of the cardiac area indicates an enlarged heart or a distended pericardium. A *depression* over the præcordium may mean a densely-adherent pericardium.

Enlargement of the veins over the chest, if slight, may be due to *mitral* or *tricuspid* disease, or to *emphysema* of the lungs; but if marked, it means pressure on the superior vena cava (if it be on both sides), or on one or other innominate vein (if it be on one side only). Such pressure is usually produced by an aneurism or mediastinal tumour, and the venous enlargement is often accompanied by œdema of the head and arms. Lastly, it must be noted that, if several very large veins can be seen

coursing over the chest and abdomen, it means that there is extreme obstruction of either the superior or inferior vena cava. For if the superior cava be greatly obstructed, the superficial veins of the chest and their epigastric anastomoses become dilated, and let the blood pass downwards into the iliac veins, and so by the inferior cava to the heart. And if the inferior cava be greatly obstructed, the blood passes from the iliac veins upwards to the superior cava by the same anastomoses. If the dilated veins on the abdomen be compressed by a band round the waist, the direction in which the blood in them is flowing can be detected by observing whether the blood is dammed up above or below the compressed point. If the blood be travelling upwards, the inferior cava is obstructed; if downwards, the superior.

The bulging of an aneurism may be visible on the chest-wall.

2. *Palpation*.—(a) As regards the *apex beat* of the heart, it is necessary to note its position and force—whether it be localized or diffuse, and whether it be accompanied by a *thrill*.

The *apex beat* is normally in the fifth interspace, about one and a half inches below and three-quarter inch inside the left nipple. It may be displaced to the *left* by hypertrophy or dilatation of the left ventricle (in which case it is usually lower than normal); by pericardial effusions (in which case it is usually higher); by the pressure of pleural effusions, hepatic abscesses, aneurisms, etc., on the right side; by the traction of pleuritic adhesions on the left side; or by the presence of a large cavity in the left lung.

The apex beat may be displaced to the *right* in consequence of effusions in the left pleura, adhesions

in the right pleura, tumours on the left side, or a cavity in the right lung. It may be displaced *upwards* by any enlargement of the abdomen, and *downwards* by an aneurism or tumour above it.

In rare cases the heart is situated congenitally on the right side, and the liver on the left.

If the apex beat be *forcible* and *heaving*, it means hypertrophy of the left ventricle. If it be *diffuse*, *i.e.*, felt over a large area, it means dilatation of the ventricles or pericardial effusion. If it be *feeble* or *absent*, it means degeneration of the heart muscle, pericardial effusion, or great overlapping of the heart by an emphysematous lung.

If the chest-wall be drawn in when the ventricle contracts, it means an adherent pericardium with much fibrous tissue between the parietal layer and the chest-wall.

Irregularity of the heart's action may be detected by palpation of the apex beat or pulse. It may be due to mental emotion, or to disease of the nerve-centres, or to uncompensated (see p. 77) valvular disease (especially mitral stenosis), or to a densely-adherent pericardium.

In some cases a vibration or *thrill* can be felt by placing the palm of the hand lightly over the apex beat. The thrill is most often præ systolic in time, and indicates mitral stenosis.

(b) At the *epigastrium* well-marked pulsation means an enlarged right ventricle, such as follows mitral disease or old-standing lung disease, particularly emphysema. A præ systolic *thrill* in this position should mean tricuspid stenosis.

(c) At the *base* of the heart a thrill means aortic stenosis, or an aneurism near the aortic valves.

(d) The expansile pulsation of an aneurism may

be felt in various parts of the chest, and it may be accompanied by a thrill.

(e) Pulsation of the *veins of the neck* can sometimes be seen and felt. If systolic, the pulsation means tricuspid regurgitation; if diastolic, it means a very adherent pericardium. A *thrill* over these veins is common in anæmia.

3. Percussion.—(a) The normal area of cardiac dulness is bounded on the right by a line drawn along the left border of the sternum from the fourth costo-sternal articulation downwards, and on the left by a slightly-curved line (with the convexity to the left) drawn from the same point to the apex beat. Below the dulness merges into that of the liver. At the edge of this area there is a zone about half an inch wide of partial cardiac dulness, where the heart is overlapped by the lung. The area of partial dulness increases at the end of a deep inspiration, and becomes less at the end of a forced expiration.

The area of cardiac dulness is *increased* when the heart is dilated or hypertrophied, and when the pericardium is distended by fluid. It appears to be increased when aneurisms, mediastinal tumours, and pleuritic effusions touch the heart's area. It is *diminished* when the lung overlaps the heart as in emphysema, and when the heart becomes small from atrophy.

(b) Dulness may be produced in various parts of the chest by aneurisms.

4. Auscultation—The Normal Heart-sounds.—Normally, two sounds are heard. The first is a dull sort of sound, and is synchronous with the systole of the ventricles and the closure of the mitral and tricuspid valves. It is loudest at the

apex of the heart. The second sound is shorter in duration and sharper in quality; it immediately succeeds the first sound, and it is synchronous with the closure of the aortic and pulmonary valves. It is loudest at the base. After the second sound there is a short pause, during which nothing is heard. The diastole of the heart takes place during the second sound and the pause.

In disease abnormal sounds may be produced (a) within the heart, (b) in the pericardium, (c) in the great vessels.

(a) **Abnormal Endocardial Sounds.**—The heart-sounds may be louder than normal, or less loud than normal, or *reduplicated*, or *abnormal in quality*, in which case they are called *bruits*.

In excited action from any cause the heart-sounds become louder. Apart from excitement, an accentuated, *i.e.*, a loud, first sound means hypertrophy of one or both ventricles. Accentuation of the second sound means *high arterial tension*, because in that condition the blood recoils on the valves with great violence as soon as the ventricular systole is over. If the accentuated second sound be loudest over the aortic valves (see below), it means high tension in the systemic arteries, such as occurs in kidney disease, extensive atheroma, etc. If it be loudest over the pulmonary valves (see below), it means high tension in the pulmonary vessels, such as occurs in mitral disease and in chronic lung affections, especially emphysema.

The heart-sounds are *less loud* if the heart be covered by pericardial effusion or emphysematous lung. The first sound is feeble, or perhaps inaudible, in fatty degeneration or atrophy of the ventricular walls.

Either sound may be *reduplicated* ; *i.e.*, there may be two normal first sounds, or more rarely two normal second sounds. Reduplication of the first sound occurs under such opposite conditions as hypertrophy of the left ventricle in renal disease, and weakening of the left ventricle in exhausting fevers. It therefore looks as if an inequality in the strength of the ventricles, or in the resistance they meet with in their respective arteries, prevented them from contracting simultaneously.

Bruits or murmurs are abnormal endocardial sounds. If they are due to valvular disease they are called *organic*, and if not, *functional*. The actual sound may be loud or soft, short or prolonged, or blowing, humming, musical, etc., in quality.

Organic bruits are produced by the blood as it rushes through an obstructed valve, or comes back through an incompetent valve. Consequently they are produced at the valves, and for each valve there is a spot on the chest-wall at which sounds made at that valve are best heard. Thus, the bruits (or accentuations, see above) which are made at the *aortic* valve are best heard at the *third right costo-sternal articulation*, those made at the *pulmonary* valve at the *third left costo-sternal articulation*, those made at the *mitral* valve at the *apex beat*, and those made at the *tricuspid* at the *base of the ensiform cartilage*.

Further, the blood-current which makes the noise at the valve carries the noise with it for a certain distance, so that endocardial murmurs are conducted in definite directions from the valve.

These points will be exemplified under valvular disease of the heart.

If a bruit is synchronous with the first sound it

is called *systolic*, and if synchronous with the second sound it is called *diastolic*, although it only occurs at the first part of the diastole. In both these cases the bruit *replaces* the normal heart-sounds. A bruit may also be heard during the pause. It is, then, most often heard just before the first sound, and is synchronous with the systole of the left auricle. It is therefore called *præsystolic* or *auricular systolic*.

Functional Murmurs.—These are heard in anæmia, and their cause is obscure. They are systolic in time, and are most common over the pulmonary artery, but they may be heard at the apex. The pulse is not affected as in valvular disease, and the other signs of anæmia help to show that the murmur is functional and not organic. In some cases of chorea the systolic bruit at the apex is functional.

(b) **Pericardial Sounds.**—These are called *frictions*, and are produced by the rubbing together of the roughened surfaces of the pericardium during the auricular and ventricular systoles, and during the diastolic dilatation of the heart. The sounds may therefore be triple, double, or single in number, according as the roughening of the pericardium is universal or localized, and in time they may be præsystolic, systolic, or diastolic, or all three. For their other characteristics see Pericarditis.

(c) **Murmurs in the Great Vessels.**—Over an *aneurism* a blowing systolic bruit is usually heard. In *anæmia* a systolic murmur can usually be obtained over the arteries in the neck, and a continuous humming-top bruit (*bruit de diable*) over the veins of the neck. Normally firm pressure on any large artery with the stethoscope may bring out a systolic murmur.

Syncope—Faintness.

The sensation of faintness can scarcely be described, but it is something like nausea, with a sinking feeling at the epigastrium. There is pallor of the face, a cold perspiration, and a weak pulse. If it goes further there is loss of vision, and then unconsciousness. If the patient be standing, he will fall to the ground.

The symptoms are due to anæmia of the brain, which is in turn due to failure of the heart's action.

The Causes of syncope are mental emotion (in which case the attack soon passes off), anæmia, Addison's disease, diabetes, fatty heart, extreme aortic stenosis, uncompensated aortic regurgitation, pericarditis, and loss of blood by hæmorrhage. In anæmia the poor quality of the blood and the weakness of the heart unite in bringing on the attack. In extreme aortic stenosis the blood cannot get to the brain. In the other cases the weakness of the left ventricle is chiefly at fault, though exertion or mental emotion usually precipitates the attack. In pernicious anæmia and in Addison's disease the attacks of syncope are prolonged and frequent, and death usually occurs in one.

Treatment.—Place the patient on his back; try to get him a free supply of fresh air, and give him ammonia to inhale. Cold water on the face is also efficacious. If he can swallow, give him brandy. For repeated attacks give strychnia, iron, and arsenic. In syncope from hæmorrhage transfusion may save the patient.

Abnormalities in the Heart's Action.

Palpitation.—In this condition the heart's action is rapid, often powerful, and sometimes irregular.

Causes.—Mental emotion, reflex irritation from the stomach, excessive smoking, uncompensated valvular disease of the heart (particularly aortic regurgitation), and exophthalmic goitre. Whenever the heart is weak (*e.g.*, in anæmia, fatty disease, etc.) exertion may cause palpitation.

Treatment.—Try to find the cause, and treat that. In particular, look for and treat dyspepsia and valvular disease.

Tachycardia.—This means rapidity of the heart's action. It may be due to mental causes, uncompensated valvular disease, weakness of the heart muscle, pericarditis, exophthalmic goitre, pyrexia, and sometimes organic brain disease.

Bradycardia.—This means slowness of the heart's action. It may be due to apoplexy, compression of the brain or other organic brain disease, pressure irritating the vagus nerve, jaundice, defervescence after a long fever.

Irregularity of the Heart's Action may be due to uncompensated valvular disease, to mitral stenosis, even when compensated, to pericarditis and adherent pericardium, to disease of the myocardium, and to mental emotion or organic brain disease, *e.g.*, meningitis.

The Pulse.

The points to be observed about the pulse are the number of beats per minute, the strength of the beats, their rhythm, their size, the amount of diastolic tension, the tension, and the thickness of the arterial wall. It may be necessary to compare the radials on the two sides, as in cases of thoracic aneurism one may be smaller than the other, or its beat may be a little later. It is sometimes necessary to examine other arteries besides the radial.

The normal *pulse-rate* varies from 65 to 75. It is quicker in children. Before counting the pulse, time must be allowed for any agitation on the part of the patient to subside. It may be necessary to compare the pulse-rate with the respiration-rate. The ratio should be 4 to 1, but in acute pneumonia it may be 2 to 1. In fevers the increase of the pulse-rate is usually proportionate to the increase of temperature, but in general tuberculosis the ratio may be disturbed. For alterations in the rate see Tachycardia and Bradycardia.

It would take too long to enumerate all the conditions which make the pulse abnormally *strong* or abnormally *weak*. As regards heart affections, the strength of the pulse usually depends on that of the left ventricle; but if there be serious aortic stenosis or an obstruction in the main arteries or in the pulmonary circulation, the heart may be acting violently while the pulse is weak. If the pulse cannot be felt at all, it must be remembered that the radial artery may be abnormally situated.

An *irregular* pulse usually goes with an irregular action of the heart (which see), but the pulse is sometimes *intermittent* while the heart is regular, because some contractions of the heart are too weak to drive the blood as far as the radial artery.

The size of the pulse is represented by the height of the sphygmographic wave, but it can be fairly well appreciated by the finger. Roughly speaking, it depends on the quantity of blood which is put into the aorta at each ventricular contraction. Consequently, when the left ventricle is dilated and hypertrophied the pulse is large, while in mitral disease the pulse is small, because the left

ventricle can only put a small quantity of blood into the aorta.

An *unequal* pulse is one in which all the beats are not of the same size, *i.e.*, the pulse-wave is not always of the same height. It usually indicates mitral disease, and must not be confounded with an irregular pulse.

The *dicrotism* of the pulse is increased when the tension in the arteries is very low, as in exhausting diseases. If the aortic valves are incompetent, dicrotism is lost.

The arterial tension is high in kidney disease, chronic malaria, and extensive atheroma. Increased tension must be distinguished from thickening of the arterial wall, although they often co-exist. For instance, in acute nephritis the arterial tension is high, but the arterial wall is not thickened, while in chronic nephritis the tension keeps high but the wall is thickened. In exhausting diseases the tension is low, and the pulse is said to be *compressible*.

A *locomotor* pulse, or, rather, a locomotor artery, means that the artery moves from side to side at each pulse. It is best seen in the lower part of the brachial artery, and it is produced by atheroma, which makes the artery tortuous, and by aortic regurgitation, which makes the blood-current jerky. The most marked locomotor arteries occur when both these factors are present, but either may produce a slight degree of lateral movement in the artery.

The phenomena of *back-pressure* are described with valvular disease.

Pain is not a marked symptom in heart disease, unless acute dilatation of the heart is occurring.

Pericarditis is painful, and most intrathoracic aneurisms cause great pain by pressing on the bones, etc. Angina pectoris is a most painful affection. Præcordial pain is often due to intercostal neuralgia or rheumatism, or to dyspepsia.

ACUTE PERICARDITIS.

Causes.—Acute rheumatism, scarlet fever (and the other fevers sometimes), septicæmia, uræmia (Bright's disease), cold, extension of inflammation from neighbouring parts, perforation of the pericardium by a wound or by the bursting of an abscess, cancer, tubercle.

Pathological Anatomy.—In simple cases the endothelium becomes shed, and an exudation occurs which coagulates and deposits fibrin on the pericardial surfaces, while more or less serum collects in the cavity. Granulation tissue usually develops beneath the fibrin, and leads to the formation of adhesions (see Pleurisy).

If the case become chronic, the granulation tissue may grow into the myocardium, producing a chronic interstitial myocarditis; and when the granulation tissue becomes fibrous, the muscular fibres may be compressed and destroyed. On the other side, dense masses of fibrous tissue may form between the pericardium and chest-wall, a condition which is called fibroid mediastinitis.

In septic cases pus forms in the pericardial cavity.

Symptoms and Physical Signs.—The disease begins with dyspnœa and præcordial pain and tenderness. There is fever, and the pulse is very quick, and may be irregular. At this stage friction-sounds are heard.

The characteristics of pericardial frictions are:

- (1) They are superficial and rubbing in character.
- (2) They are not necessarily best heard at the points on the chest-wall corresponding to the valves, but are most often heard at the base of the heart.
- (3) They are heard over a limited area, and are not conducted in definite directions like endocardial bruits.
- (4) They vary from time to time, being sometimes audible and sometimes not.
- (5) They may become louder on pressure with the stethoscope.
- (6) They may be systolic, diastolic, or præ systolic, and are often all three, so that a triple sound, compared to a canter, may be heard. When systolic or diastolic, they do not replace the heart-sounds, although they are synchronous with them; for instance, a systolic rub and a normal first sound may be heard together, but quite distinct one from the other to the ear.

To distinguish peri- or exo-cardial sounds from endocardial bruits, it must be remembered that the latter are loudest at certain points on the chest-wall, are conducted in definite directions from those points, and replace the normal heart-sounds. They are also much more constant, except in septic endocarditis.

When the fluid accumulates in the pericardium, the area of cardiac dulness becomes increased, and the shape becomes altered to that of a triangle, the apex of which is above and the base below. This triangular shape enables one to distinguish pericardial effusion from a dilated heart, for in the latter the dull area is quadrilateral. At the same time, the apex beat is displaced upwards and to the left, while in a dilated heart it is lower than normal and very diffuse.

The heart's action is now much embarrassed, and in consequence the dyspnœa may be great; but the frictions may be less loud or absent, and the heart-sounds may be distant. If the amount of fluid be great, the præcordial dulness may reach to the first rib, and the lower lobe of the left lung may be compressed against the chest-wall, so that dulness, tubular breathing, and bronchophony may be obtained at the left base (see p. 45). The patient may die at any moment from failure of the heart. Renal and, of course, septic cases are the most fatal. When the fluid becomes absorbed, the frictions become loud again, and disappear as recovery takes place.

Treatment.—Apply poultices or leeches to the præcordium to relieve the pain. Morphia may be given at first with the same object, and it may be combined with diaphoretics. When the fluid is abundant the morphia must be stopped, and strychnia, digitalis, ether, and brandy may be given. Diuretics and hydragogue purgatives may be tried to assist the absorption of the fluid, and large blisters to the præcordium are also used for this purpose, while paracentesis of the pericardium might be performed in extreme cases. Iodide of potash is useful as an absorbent when the acuter symptoms have subsided.

By **Hydropericardium** is meant an effusion of simple serum into the pericardium, such as occurs in general dropsy from kidney disease, cardiac valvular disease, etc. After death it is distinguished from pericarditic effusion by the absence of fibrin, and during life by the absence of frictions.

Adherent Pericardium.—This condition results from pericarditis. The physical signs are irregular

heart's action, systolic recession of the apex beat, diastolic collapse of the veins in the neck, and the pulsus paradoxus, *i.e.*, a pulse which becomes rapid and small during inspiration. Further, the area of præcordial dulness does not vary with respiration, as it should. There are no abnormal sounds to be heard. The above symptoms, however, indicate fibroid mediastinitis rather than mere adhesion of the pericardium. Persons with an adherent pericardium often die suddenly, but all the symptoms of a valvular disease may have been present *except the bruits*.

ENDOCARDITIS.

Causes.—Acute endocarditis may be (1) simple, in which case it may be due to rheumatic fever, scarlet fever, and sometimes the other fevers; or (2) septic, in which case it is due to pyogenic bacteria, although the part of the body at which the bacteria enter the blood cannot always be found.

Chronic endocarditis usually follows an acute attack, but may occur as a primary affection, and be due to gout, kidney disease, syphilis, alcoholism, chronic rheumatism, or prolonged excessive muscular exertion, in which case it is associated with chronic endarteritis.

Pathological Anatomy.—1. In Simple Endocarditis, the vitality of the endocardium becomes impaired, so that the endothelial cells on the *valves* become detached at the place where the valves come in contact with each other when they close. The right side of the heart is very seldom affected. As soon as the endothelium is detached, leucocytes cling to the raw surfaces, and fibrin (from coagula-

tion of the blood) is deposited on them. These little masses of fibrin and leucocytes are called *vegetations*. Sometimes the vegetations become absorbed; but more often granulation tissue grows in the valve, so that the vegetation comes to consist of fibrin on a base of granulation tissue. When the granulation tissue becomes fibrous tissue and contracts, the valves become white, opaque, thick, distorted, and rigid. They may also adhere to each other. These changes lead to valvular disease.

The growth of new fibrous tissue indicates that the acute endocarditis has become **Chronic**, but the same growth may occur (from the causes given above) without any acute stage, and it leads to the same thickening, distortion, etc. In chronic cases, also, the valves may become calcareous.

2. In **Septic Endocarditis** vegetations also occur, but they are larger and softer, and are crowded with micrococci. The cocci cause great destruction of tissue, so that the valve may become perforated, or may even hang in strips. Aneurisms of the valves may also occur. The lesions are not confined to the valves, and soft masses of fibrin may be found all over the endocardium. The right heart may be affected as well as the left. This affection has been called *ulcerative endocarditis*, because of the extensive detachment of endothelium. As the vegetations are so soft, they are easily broken up, and the pieces which are carried away as emboli by the blood-stream become impacted in the small arteries of various organs, and lead to the formation of infarctions which become abscesses.

The **Symptoms of Simple Endocarditis** are those of valvular disease.

Symptoms of Septic Endocarditis.—(1) There is remittent fever, which is often high, and associated with recurrent rigors and sweatings. Many cases occur during the course of old-standing valvular disease, and so the onset may be insidious. (2) Endocardial bruits can usually be heard, but they are very variable, because the masses of fibrin which cause them are soft, and are constantly being broken up. They may be accompanied by the other symptoms of valvular disease, such as dropsy, etc. (3) The symptoms of the *typhoid state*, such as delirium, coma, dry brown tremulous tongue, subsultus tendinum, etc., are usually present, and may be so marked as to cause the case to be taken for typhoid fever. Jaundice may also be present. (4) Symptoms due to emboli are almost certain to occur. If the emboli lodge in the *spleen*, there is a sudden enlargement of that organ, which becomes painful and tender; if in the *kidney*, there is a sudden pain in the loins, followed, perhaps, by albuminuria or hæmaturia; if in the motor region of the *brain*, there is sudden hemiplegia; and if in the *lungs* (in which case thrombi must have formed in the right heart), there is a sudden pain in the side, followed by dyspnœa, hæmoptysis, and crepitation, or even the physical signs of consolidation over the infarction. Emboli may also block the main artery of a limb, and cause a stoppage of the pulse, and then gangrene. The impaction of emboli usually causes a fresh rigor, and an exacerbation of the fever. These cases are almost invariably fatal, and no special treatment can be recommended.

VALVULAR DISEASE OF THE HEART.

Causes.—1. **Endocarditis.** The presence of vegetations on the valves, and the distortion, inflexibility and adhesion of the valves which result from endocarditis, may narrow the orifice of the valve, producing what is called *stenosis*, and may prevent the accurate closure of the valves, and so allow of *regurgitation*. Rheumatism, etc., cause valvular disease by producing endocarditis.

2. **Dilatation of the Valvular Orifices.**—This may be so great that the valves cannot close them (although the valves are normal), and, consequently, regurgitation occurs. Thus, an aneurism may stretch the aortic orifice, and a dilated left ventricle the mitral.

3. **Rupture.**—The aortic valves may rupture during a great muscular effort; but probably in such cases the valve is already diseased.

4. **Congenital Malformation.**—See p. 88.

Symptoms and Physical Signs of Valvular Disease of the Heart.—The great indication of valvular disease is the presence of endocardial bruits, the nature of which has been described at p. 64. In every case it is necessary to note the time at which the *bruit* is heard, the place at which it is loudest, and the direction in which it is conducted. The presence or absence of a *thrill* must next be noted, and the time at which it occurs. Then the pulse must be examined, and then the back-pressure symptoms. Before going further, the nature of the back-pressure must be explained.

Back-pressure and Compensation in Valvular Disease.

In stenosis of a valve the blood cannot pass through the heart freely, and in incompetence of a valve the blood passes back again after it has gone through the valve, so that in both cases the circulation is impeded. Consequently the blood becomes dammed up behind the affected valve, and a venous engorgement of the various organs takes place and gives rise to pathological changes and corresponding symptoms, which are called back-pressure symptoms. These are given in detail under Mitral Regurgitation. Now, in an otherwise healthy person (after a transient period of dilatation) the muscular wall of the heart-cavity behind the valve becomes hypertrophied, and may be sufficiently strong to keep the blood going onward through the valve, in spite of the stenosis or regurgitation, and then no back-pressure symptoms occur. In such a case the valve lesion is said to be *compensated*. But if the hypertrophy be not kept up, the back-pressure symptoms appear, and the valve lesion is said to be *uncompensated*.

Back-pressure symptoms are, therefore, the great indication of imperfect compensation; but a deficiency of blood in the arteries (that is, in the vessels in front of the diseased valve) also indicates imperfect compensation, because it shows that enough blood is not getting through the heart. Thus, the attacks of syncope which occur in disease of the aortic valves, and which are due to anæmia of the brain, are signs of non-compensation. Irregularity of the heart's action when there is valvular disease also usually means want of compensation.

If the muscle of the heart become fatty or otherwise weak, the blood may not be propelled from the heart properly, and may consequently tend to be dammed up in the venous system, and to produce all the back-pressure phenomena, although the valves are normal.

The symptoms, etc., of disease of each valve may now be considered.

Mitral Regurgitation.

There is a *systolic* bruit, loudest at the *apex*. It is conducted into the left *axilla*, and to the *angle of the scapula*, because the regurgitating current which makes the bruit goes into the auricle, and therefore carries the bruit towards the back. A *thrill* is rare, but if felt is *systolic*. The pulse is *small*, and may be *unequal*. If the valvular defect be compensated, the left auricle becomes hypertrophied, and no other symptoms occur. But if it be uncompensated, the heart's action may become irregular, and the following back-pressure phenomena are present :

The **Lungs** become *congested*, which causes *dyspnœa*, which is worse on exertion. If the back-pressure gets worse, the lungs become *œdematous*, which increases the *dyspnœa*, and gives rise to *diminished resonance* and *non-consonating crepitation* at the bases. Or sometimes *hydro-thorax* occurs, causing *dulness* and *loss of breath-sounds*, and of *vocal resonance* and *fremitus*. In the worst cases the lung capillaries rupture, giving rise to *hæmoptysis*. The *dyspnœa* may amount to *orthopnœa*.

The tension in the **Pulmonary Artery** is raised, which causes an *accentuated second sound* at the *pulmonary valve* (see p. 64).

The **Right Ventricle** becomes *hypertrophied* (from having to work harder to get the blood through the congested lungs), and later on *dilated*. This causes an *increase of præcordial dulness* towards the right, and *epigastric pulsation*.

The **Right Auricle** also becomes *hypertrophied* and *dilated*, but this does not cause any special symptoms unless the *tricuspid orifice* becomes so dilated that the valves cannot close it. In such a case *tricuspid regurgitation* will occur (which see).

The hepatic veinlets in the **Liver** become distended, so that the liver becomes large and smooth, and at times painful and tender. In bad cases *ascites* follows the congestion of the liver, and *jaundice* may set in.

The **Stomach** becomes congested, and often catarrhal. This causes *dyspepsia*, and sometimes *hæmatemesis*.

The **Spleen** is congested at first, but soon becomes *small and fibrous*.

The **Kidneys** are large and congested, so that the *urine* is scanty, high-coloured, and albuminous. The high colour and the comparative absence of *casts* distinguish cardiac urine from the albuminous urine of Bright's disease.

The **Subcutaneous Tissue** becomes *œdematous*. This dropsy begins in the *legs*, and extends eventually to the trunk and to all the serous cavities. The escape of serum through the capillary walls (which constitutes the dropsy) is due more to the impaired nutrition of the capillaries on account of the stagnant impure blood in them than to their mere over-distension with blood. The dropsy of the feet is often the earliest back-pressure symptom, because the circulation there is the most difficult to carry on, and therefore feels the strain first.

It must never be forgotten that all the above may be symptoms of mitral regurgitation. In some old cases of mitral regurgitation the *left* ventricle becomes hypertrophied, because the dilated left auricle puts so much blood into it at each contraction that the ventricle has to work harder to expel it. But as most of the blood goes back into the auricle, this hypertrophy does not help the patient much.

Post-mortem.—There may be vegetations on the valves, or the orifice may be simply dilated (the normal circumference is about four inches), or the valves may be too rigid to shut properly, or the chordæ tendineæ may be too short (from new

fibrous growth) to allow them to meet. The legs are dropsical, and there is clear serum in the pericardium, pleuræ, and peritoneum. The lungs are congested and œdematous, and perhaps hæmorrhagic. In old cases the lungs become fibrous and shrink, a condition which is called brown induration. Both auricles and the right ventricle are dilated, and have walls which are thickened from hypertrophy. The liver is large and smooth, and on section shows abundant black spots (which are dilated veins) surrounded by yellow rings (which are composed of fatty liver-cells). In old cases it may be small, hard, and red, a condition called red atrophy. The spleen is small and fibrous, and the stomach and kidneys are congested. In all cases of valvular disease infarctions may be found in the various organs.

Mitral Stenosis.

There is a bruit which is usually *præsystolic*, but may be heard at any time during the pause between the second sound and the first sound. It is loudest at the *apex*, and is not conducted beyond that point, because the blood-current which makes the bruit as it passes the narrowed valve impinges against the ventricular wall near the apex. A *præsystolic thrill* at the apex is common, and the pulse is *small*, and often *irregular* and *unequal*. The back-pressure symptoms are the same as those of mitral regurgitation (which see), and the heart's action is very often irregular.

Post - mortem.—The orifice of the valve is narrowed. Sometimes the two valves become adherent at the edges to form a perfect ring,

which makes the orifice like a *buttonhole*, and the whole valve like a *funnel*. The other post-mortem appearances are the same as in regurgitation.

It may be noted that mitral stenosis is usually associated with regurgitation, because the edges of the valve are too rigid to shut properly. But regurgitation is very common without stenosis. In mitral disease death usually occurs from back-pressure, *i.e.*, from general dropsy, or from inflammation in the œdematous organs, *e.g.*, pneumonia, erysipelas in the legs, etc. It may also be noted here that three sounds are produced at the apex, in (1) mitral stenosis, (2) pericardial friction, and (3) reduplication of the first sound (see pp. 64, 71).

Aortic Regurgitation.

There is a *diastolic* bruit, loudest at the *third right costo-sternal articulation*, and conducted down the sternum, because that is the direction of the blood-current as it drops back into the ventricle after having made the bruit at the valve. A *thrill* is rare, but would be *diastolic*. The pulse is *full and collapsing*, and the arteries may be *locomotor*. The left ventricle becomes hypertrophied (to compensate for the valvular defect), so that the apex-beat is displaced to the left and downwards, the impulse is heaving and strong, and the præcordial dulness is increased. As long as the hypertrophy is kept up, no further symptoms occur, but when it fails the left ventricle becomes *dilated* from back-pressure, and *palpitation*, *irregular heart's action*, *dyspnœa*, and *attacks of syncope* (from anæmia of the brain), occur. The face is pale, and a *pulsation of the capillaries* may be observed if the skin be reddened, or if a glass slide be pressed on the inside

of the lower lip. The left ventricle may become so dilated that the mitral valve becomes incompetent.

The signs of mitral regurgitation complicating aortic regurgitation are (1) the appearance of a new bruit (systolic at the apex, and conducted to the back); (2) the pulse, which had been full and collapsing, becomes small and collapsing; (3) mitral back-pressure symptoms, such as dropsy, etc., appear.

In aortic regurgitation death occurs from sudden syncope due to failure of the left ventricle, or from the supervention of mitral regurgitation.

Aortic Stenosis.

There is a *systolic* bruit, loudest at the *third right costo-sternal articulation*, and conducted *upwards* along the arteries, because the blood-stream travels up there after having made the bruit at the valve. There is usually a *systolic thrill* at the base. The pulse, in uncomplicated cases, is *small*. The left ventricle becomes hypertrophied.

Aortic stenosis and regurgitation usually occur together, because the valves, being rigid, will neither open nor shut properly. They cause a double bruit at the base. Angina pectoris is a common complication of aortic valvular disease. A systolic murmur at the base is common in old people, and if they are otherwise well it only means slight roughening of the valve; but if they are constantly drowsy, and suffer from attacks of syncope, there is probably serious narrowing of the orifice.

Post-mortem.—The aortic valves are found to be rigid and distorted, and perhaps calcareous. The normal circumference should be about three and one-

sixth inches. The orifices of the coronary arteries may be narrowed by the thickening or calcification. This condition leads to dilatation of the left ventricle, because hypertrophy cannot be maintained without a free blood-supply. The left ventricle will be dilated and hypertrophied, and if the mitral valve be incompetent, all the pathological changes due to back-pressure (which see) may be present.

Tricuspid Regurgitation.

This occurs (*a*) when the right ventricle becomes greatly dilated from mitral disease, (*b*) in septic endocarditis sometimes, (*c*) in congenital heart disease.

It causes a *systolic* bruit, loudest at the *base of the ensiform cartilage*. The veins in the neck show a systolic pulsation.

Tricuspid Stenosis.

This may occur in septic endocarditis. It is not very uncommon in association with a *buttonhole* mitral in young persons. Such cases may be congenital. It causes a *præsystolic* bruit, loudest at the *base of the ensiform cartilage*.

If the tricuspid be the only valve affected (either by stenosis or regurgitation), the back-pressure begins in the right auricle, and the liver, etc., suffer severely; but the lungs get less blood than they should.

Pulmonary Valvular Disease.

This is very rare, except in the form of congenital stenosis of the valve.

Pulmonary stenosis causes a *systolic* bruit, loudest at the *third left costo-sternal articulation*. The lungs are anæmic, and often become tubercular. Most systolic bruits at the pulmonary valve are anæmic in origin.

The **Prognosis in Valvular Disease** depends on (a) the individual affected, (b) the valve affected, (c) the symptoms which are present. The valve itself cannot be expected to recover, and everything depends on maintaining the compensating hypertrophy.

(a) In full-grown persons the prognosis is better than in the very young and old, because they can expend extra nutrition on the heart muscle. Persons whose occupation is light and not exciting may go on for years. In persons who have any other disease, especially in the kidney or lung, the prognosis is bad. It is also bad in pregnant women and alcoholic persons.

(b) Aortic regurgitation is the worst valvular lesion, because (1) the coronary arteries can often not supply enough blood to the heart muscle; (2) it is often due to a primarily chronic endocarditis, and so comes on late in life; (3) it is often associated with atheroma of the arteries, and the arterial disease gives the heart still more work to do, so that it soon gives out. Mitral stenosis is the next worst, because the left auricle never empties itself properly, and back-pressure therefore comes on early. Mitral regurgitation is the next, and pure aortic stenosis is the least serious, probably because when unassociated with regurgitation the narrowing of the valve is slight.

(c) The appearance of back-pressure symptoms is, of course, of bad omen. In aortic disease attacks

of faintness and the supervention of mitral regurgitation indicate that the end is near, and in mitral disease inflammation of any of the œdematous organs is always dangerous.

Treatment of Valvular Disease.—The objects of treatment are : (1) to procure or maintain the compensating hypertrophy of the heart muscle ; (2) to relieve the symptoms which occur when compensation fails.

If the valvular defect be compensated (which is recognised by the absence of the back-pressure symptoms, etc.), the patient should simply live hygienically, eating and drinking moderately, taking moderate exercise, and avoiding excitement.

If there be a moderate want of compensation (evidenced by dyspnœa on ordinary exertion, slight puffiness of the feet after standing, etc.), it may be sufficient to aid the nutrition of the heart by giving iron, arsenic, cod-liver-oil, phosphates, etc., to enjoin comparative rest, and to order specially nutritious food. Stimulants should never be ordered, but the patient, if previously temperate, should go on taking what he is accustomed to.

The above rules apply to affections of all the valves, but when compensation has failed, aortic and mitral diseases must be considered separately.

In **Uncompensated Mitral Disease** (which will be recognised by the dilatation of the right ventricle, the congestion of the lungs, dropsy, etc.) the nutritive treatment must be continued, and absolute rest enjoined. But it is now necessary (*a*) to give cardiac tonics to assist the heart muscle, and (*b*) to take measures which will relieve the congested veins in all the organs.

(*a*) The best cardiac tonic is digitalis, which

increases the actual power of the ventricular contraction, and slows and makes regular the heart's action. It lengthens the diastolic period, which is especially useful in mitral stenosis, because it gives time for the blood to flow through the narrowed valve. It also raises the tension in the systemic arteries, which does no harm in mitral disease, because the left ventricle is not doing extra work, although the right ventricle is. Digitalis is also a diuretic, and so assists in relieving the venous congestion. For all these reasons digitalis is most useful in mitral disease.

Strophanthus is also a good cardiac tonic, and differs from digitalis in not raising the arterial tension so much. Five minims of the tincture in an ounce of aq. chloroform. is a good prescription. Caffeine is another cardiac tonic. Strychnia, although it has not the special action of digitalis, is an excellent stimulant to the heart.

Cardiac tonics soon lose their effect, and, therefore, should not be wasted by being given while the valve defect is perfectly compensated. A change from one cardiac tonic to another is often most beneficial.

(b) To relieve the venous congestion, and, consequently, the over-distension of the right ventricle, give *hydragogue purgatives*, such as jalap, scammony, and elaterium. These, by removing water from the capillaries of the intestine, relieve the venous congestion of the liver, stomach, etc. Also give *diuretics*, such as the acetate or citrate of potash, scoparia, squill, juniper, copaiba, etc., and particularly digitalis. These also remove a lot of water from the body. It may also be necessary to tap the pleura or peritoneum, or even the legs, so

as to drain off the serum. Finally, venesection will often relieve the right ventricle for a time, as in lung disease (see p. 4).

To sum up, a case of uncompensated mitral disease may be treated with drugs as follows: Give pulv. jalap. co., \bar{z} i., every morning, and a mixture containing tinct. digital., \mathcal{M} x.; potass. citrat., \bar{z} ss.; spir. juniper, \bar{z} i.; decoct. scopariæ ad \bar{z} i.; three times a day.

Brandy must be given if the dyspnœa be great, but only to tide over the period of non-compensation. Gin is perhaps better, as it is a good diuretic.

Some authorities recommend mercury in valvular disease.

In **Uncompensated Aortic Disease** (which may be recognised by palpitation and attacks of syncope, etc.) we cannot give digitalis, because (*a*) it lengthens the diastole, and therefore allows the blood to fall back through the incompetent valve for a long period, and (*b*) it contracts the arteries, and gives the already overtaxed left ventricle more work to do to get the blood through them. Strophanthus may be tried cautiously, because it does not raise the arterial tension so much. We have, therefore, to content ourselves with making the patient rest, and giving him strychnia, iron, etc.

Stimulants should not be given regularly, because, although they whip up the heart, the effect passes off and leaves depression. But if attacks of syncope are frequent, and the patient has to keep his bed, brandy must be given. If in these cases the left ventricle becomes very greatly dilated, and secondary mitral regurgitation sets in, then digitalis

may be tried cautiously, and purgatives, diuretics, etc., will be found useful.

There are no means of preventing endocarditis from occurring in the course of rheumatic fever. The salicylates which check the fever and the articular affection do not save the heart. But if endocarditis occur, prolonged rest must be insisted upon, even after the joints are well, and even if the bruits have disappeared, because we know that granulation tissue is present in the valves, and the more the patient moves (and thereby increases his heart's action), the more granulation tissue forms, and the more granulation tissue that forms, the more distortion of the valves will occur when it becomes fibrous and contracts.

CONGENITAL HEART DISEASE.

Causes.—(1) Maldevelopment of the heart. (2) Endocarditis during foetal life. If the heart and valves be perfectly formed, but the latter show adhesions, thickenings, etc., such as we know result from post-natal endocarditis, we conclude that endocarditis has occurred in utero.

Varieties.—(1) Patent foramen ovale, which may even amount to absence of the auricular septum, so that there is only one auricle. (2) Incomplete closure of the ventricular septum, leaving an opening which is always at the base of the heart, because the septum grows from the apex upwards. Sometimes the whole septum may be absent, so that there is only one ventricle. (3) The pulmonary orifice may be absent, or there may be two pulmonary valves only, or the valves may be adherent. Defects in the pulmonary valve are associated with (4) patency of the ductus arteriosus, in

which case the blood-current is the reverse of that in the fœtus, for the blood passes from the aorta through the ductus into the lungs, instead of from the pulmonary artery through the ductus into the aorta. Other defects are more rare, but the division between the aorta and pulmonary artery may be absent, or the aorta may come from the right ventricle, and the pulmonary artery from the left. Congenital tricuspid and mitral stenosis appear to result from endocarditis. In some cases of anæmia the aorta is congenitally small.

Symptoms.—The face, hands, and legs, are deep blue in colour, and have given the name of cyanosis, or the blue disease, to the affection. The conjunctivæ are suffused and blue, and the nose and lips are large, fleshy and blue. The ends of the fingers are bulbous and blue. The feet are almost black, and both hands and feet are always cold. The blueness is supposed to result from the mixture of venous and arterial blood. There is habitual dyspnœa, and there may be a bruit, usually at the base of the heart. A few cases of even well-marked cyanosis have been known to reach adult life.

Treatment.—Keep the patient warm, and guard against lung troubles. Sometimes back-pressure symptoms, especially dropsy, occur, and they must be treated as in mitral disease.

DISEASES OF THE MYOCARDIUM.

Hypertrophy of the Heart.—This means a thickening of the ventricular or auricular walls from increase in the number, and perhaps in the size, of the muscular fibres of the heart, and it always results from overwork.

The *left ventricle* is hypertrophied in persons who follow laborious occupations, and in athletes. Also when the arterial tension is raised, as in kidney disease, chronic endarteritis, etc. Also in any disease of the aortic valves, so long as the extra work of the muscle is not too great, and so long as it gets a free supply of blood. Hypertrophy may also follow the functional overwork which occurs from the palpitation of exophthalmic goitre, and it is present in pregnancy.

The *right ventricle* becomes hypertrophied in mitral disease, and in all chronic lung affections, from increased tension in the pulmonary artery. Hypertrophy of the *auricles* results from mitral or tricuspid disease respectively.

If a hypertrophied heart becomes dilated, there is said to be eccentric hypertrophy, and the thickening of the wall may still be evident, although it is not so great as it was. By concentric hypertrophy was meant a thickening of the ventricular wall at the expense of the ventricular cavity, but this probably does not occur.

Hypertrophy is indicated by a strong impulse of the heart, and an increase in area of the præcordial dulness. If the left ventricle be affected, the apex beat is displaced downwards and to the left, and the sphygmographic pulse-wave is high, and ascends gradually. If the hypertrophy be due to increased arterial tension, the second sound at the aortic valve may be accentuated. The right ventricle produces strong epigastric pulsation, and the high tension in the pulmonary artery which has produced the hypertrophy causes an accentuation of the second sound at the pulmonary valves (see p. 64). In both cases the first sound or the second may be reduplicated.

Dilatation of the Heart.

This occurs from weakness of the heart (as in fatty disease) or from the failure of hypertrophy, particularly in valvular disease; so that the causes of hypertrophy of either ventricle or auricle can be referred to (see above) as ultimately proving the causes of dilatation. The determining cause of the change from hypertrophy to dilatation may be an increase of the primary cause (as when a valve lesion gets worse), or a deficient supply of blood (as when the coronary arteries are narrowed), or imperfect general nutrition, as in old age, etc. When persons who have been born with or acquire a weak constitution attempt severe athletic performances, the heart cannot hypertrophy, and will dilate, sometimes sufficiently so to make the valves incompetent. Hypertrophy from renal disease does not give way for a long period.

The right ventricle may be so large as to form the apex of the heart, particularly if the previous hypertrophy has been great.

Putting aside the cases of simple weakness of heart muscle, the **Symptoms** of a dilated heart are those of uncompensated valvular disease, and vary according as the aortic or mitral valve is most affected. The area of heart dulness is increased and square in shape, the impulse is diffuse and feeble, and the pulse exhibits the characteristics of the valvular affection.

The **Treatment** is the same as that of valvular disease.

Myocarditis.

Acute myocarditis usually complicates septic endocarditis or some other form of septicæmia, and

has no symptoms of its own. The heart muscle is soft, and may show abscesses, or, microscopically, infiltration with leucocytes and fatty muscular fibres. Chronic myocarditis usually complicates adherent pericardium (see p. 70).

Fatty Disease.

In *fatty infiltration* the connective tissue of the myocardium is distended by fat globules, which separate the muscular fibres. It is most common in general obesity.

In *fatty degeneration* the muscular fibres lose their nuclei, and their protoplasm is converted into oil, which can be seen inside the remains of the fibre. It occurs conspicuously in pernicious anæmia, in which case a minute yellow striation or mottling of the myocardium can be seen with the naked eye, especially in the muscoli pupillares. It may also be due to prolonged pyrexia, phosphorus-poisoning, alcoholism, etc., and may follow stenosis of the coronary arteries, or failure of nutrition after hypertrophy.

Symptoms.—Fatty degeneration causes weakness of the heart, and has in time the same effect as valvular disease (see p. 78). Consequently all the symptoms of aortic disease (drowsiness, faintness, etc.), and all the symptoms of mitral disease (dyspnœa, dropsy, etc.), may be present. But the bruits of valvular disease and the pulse-changes are absent. Usually the impulse is weak or absent. The first sound is weak at the apex, and absent at the base; and the second sound is audible as a sharp, but not really loud, click at the aortic valves. The area of heart-dulness may be increased. The pulse is often intermittent.

Treatment.—Give iron, arsenic, and strychnia, and attend to hygienic considerations. If dropsy set in, digitalis may be given with caution, and the treatment of uncompensated mitral disease adopted.

OTHER AFFECTIONS.

Cancer of the Heart is rare. It usually grows in the pericardium, and causes the symptoms of pericarditis. The pericarditic effusion is blood-stained.

Tubercles also usually are limited to the pericardium, but may be found in the myocardium. There may be tubercular pericarditis.

Syphilis may cause chronic endocarditis, and consequent valvular disease. Gummata have been found in the myocardium.

Hydatids have been found in the myocardium, and might produce peri- or endo-carditis by bursting in either direction.

In **Lardaceous Disease** the arterioles of the heart show the lardaceous change distinctly.

Brown Atrophy of the Heart consists in a shrinkage of the muscular fibres, the protoplasm of which becomes replaced by brown pigment granules. The symptoms are like those of a fatty heart, and the patients are usually old.

ANGINA PECTORIS.

Symptoms.—There is a sudden excruciating pain at the heart. The pain extends to the left shoulder and down the arm. There is a sense of oppression, and the patient thinks he is dying. The face is pale, there is some dyspnœa, and often a cold perspiration. There are no physical signs (unless

heart disease is also present), but the heart's action may be irregular, and the pulse is tense. There is no præcordial tenderness. The paroxysms may recur frequently or at long intervals, and they are brought on by emotion or by exercise, especially by going up hill. The disease is most common in elderly men.

Pathology.—In most cases there is a *spasm of the muscular coat of the arteries* during the attack, and as the left ventricle has to drive the blood through the narrowed arteries, its contractions become painful, just as the intestinal contractions, which are normally painless, become painful when they work against an obstruction. This explains the pain; but the cause of the arterial spasm is very obscure. In some cases no lesion is found after death, and we have to consider the spasm as a neurosis, *i.e.*, a functional nervous disease. But in most cases there is disease at the aortic valves, especially a great thickening or calcification of the valves, which spreads to the coronary arteries and the adjacent heart muscle. In these cases it is possible that the cardiac plexuses of sympathetics may be irritated by the thickening, and may induce the arterial spasm.

In cases where no arterial spasm can be demonstrated, it is suggested that anæmia of the heart may make its contractions painful, and stenosis of the coronary arteries would, of course, produce anæmia.

Treatment.—Inhalation of a few drops of nitrite of amyl relieves the pain by relaxing the arterial spasm. It acts very quickly, but the effect soon passes off. Nitro-glycerine has the same effect, but its action lasts longer, and it is therefore better

for cases in which the paroxysms recur frequently. The dose is $\frac{1}{100}$ grain in a tabloid or in solution. The rest of the treatment consists in looking after the general health, and warning the patient against the exciting causes.

Prognosis.—If there be no valvular lesion the prognosis is good.

DISEASES OF THE ARTERIES.

ATHEROMA—CHRONIC ENDARTERITIS.

Pathology.—In this disease new round cells appear in the subendothelial connective tissue of the bloodvessels. In the arterioles this infiltration may be uniform, but in larger arteries it occurs in patches. Probably there is some preceding molecular degeneration of the intima or media, at the place where these patches form. They are common at the places where branch arteries leave a main stem. If the patches be small, the new subendothelial cells develop into granulation tissue, and later into fibrous tissue. This usually occurs in the arterioles. In the larger patches the cells next to the media die and become caseous, while those next to the endothelium become fibrous, so that fibro-caseous patches are formed. The patches are slightly raised, flattened on the top, and more yellow and opaque than the rest of the arterial wall. The aorta and the cerebral arteries exhibit these so-called atheromatous patches most frequently. The artery is no weaker at the site of the patch, but is probably weaker just around it. But the patches make the artery less elastic, and narrow the smaller arteries, so that they impede the circulation. In some cases the new cells become fatty, instead of developing to granulation tissue. Then the endothelium is undermined by a

fatty pulpy mass, which is called an *atheromatous abscess*, and when the endothelium breaks and the pulp escapes, an *atheromatous ulcer* is left, which constitutes a weak spot in the arterial wall, at which the blood-pressure may develop an aneurism.

In other cases the new cells either before or after they have become fibrous are impregnated with lime salts, making the arterial wall calcareous.

Fatty degeneration of the muscular coat is often present, and the vasa vasorum are surrounded by leucocytes.

Causation.—The disease is always present in old people. In younger people it results from laborious occupations, kidney disease, gout, alcohol, syphilis, and anything that makes a man older than his years. It is not so common in women.

In syphilis the round cells may appear in the muscular coat as well as beneath the endothelium; and near gummata, the endothelium itself may proliferate, so that the vessels become extremely narrowed.

ANEURISM.

Pathology.—An aneurism is a localized dilatation of an artery, and is due to (*a*) weakening of the arterial wall, and (*b*) increased blood-pressure inside the artery. The latter alone would not cause aneurism.

The weakening of the arterial wall may be due to (1) chronic endarteritis, especially when the new subendothelial cells become fatty and form the atheromatous ulcer; (2) degeneration of the muscular coat; (3) laceration of the inner coat by calcareous spiculæ, etc.; (4) acute inflammation of

the inner coat, due to a septic embolus impacted in contact with it; (5) a wound from without, especially when the adventitia and middle coat are alone cut or torn. The increased arterial tension is most often due to prolonged muscular effort, but pressure on an artery may cause an aneurism at a point just above the compression.

The causes of atheroma, and particularly syphilis, are, of course, also causes of aneurism. Aneurisms are rare in women, and are most common in middle-aged men in whom pathological atheroma has occurred at an age when they still make strong muscular efforts.

Varieties.—In a fusiform aneurism the lumen of the artery is dilated to form a more or less oval cavity.

A sacculated aneurism stands out as a bulge from one side of the artery, and its cavity communicates with the lumen of the artery by a lateral hole in the arterial wall. The aneurismal wall may be made up of one or two of the arterial coats.

In a dissecting aneurism the cavity is an elongated one, and situated in the muscular coat of the artery. It results from rupture of the inner coat, whereby the blood reaches the middle coat and travels up the arterial wall between the muscular fibres. It usually breaks into the artery again at some point higher up.

In a diffuse aneurism the arterial wall is quite absent, and the aneurismal wall is made up of the surrounding tissues. It often follows rupture of one of the other kinds.

There are other kinds of aneurism, but they are chiefly of surgical interest.

Aneurism of the Thoracic Aorta.

Symptoms.—These are of two kinds :

(1) *The aneurismal physical signs*, which are expansile pulsation, bruit, thrill, and dulness on percussion.

(2) *The symptoms due to the pressure of the aneurism on the surrounding parts.* These may also occur with solid mediastinal tumours.

Aneurisms may occur in any part of the thoracic aorta, but three situations may be taken as examples :

1. **Aneurisms of the First Part of the Arch** usually bulge forwards, so that the aneurismal signs appear at about the second or third right costal cartilage. The sternum and ribs (but not the costal cartilages) become eroded, and cause pain. Bulging to the right compresses the superior vena cava, giving rise to œdema of the head and neck and both arms, and sometimes to great distension of the abdominal veins (p. 60). Bulging backwards compresses the root of the lung, causing feeble breath-sounds from pressure on the bronchus, and œdema of the lungs, or hydrothorax, from pressure on the veins. The heart is often displaced to the left and downwards. Death may occur from rupture through the skin, into the pericardium, etc., or from pressure on the lung.

2. **Aneurisms of the Transverse Arch.**—The aneurismal signs appear behind the manubrium sterni; but, except the bruit, they may be absent unless the sac bulges forwards. The left recurrent laryngeal is paralyzed from compression, so that there is aphonia, and the left vocal cord lies motionless midway between abduction and adduc-

tion. Bulging backwards compresses the trachea, causing dyspnœa and a barking cough. Bulging upwards compresses the left innominate vein, causing œdema of the left side of the face and left arm. Bulging to the left compresses the œsophagus. The arteries which come off from the arch are often involved, so that the two radial pulses may be unequal. Death may occur from pressure on the trachea, or rupture into the trachea, or elsewhere.

3. **Aneurisms of the Descending Aorta.**—The aneurismal signs may be long absent, but, as a rule, eventually appear in the back. Compression of the œsophagus, causing dysphagia, and of the thoracic duct, causing starvation, is common. Erosion of the vertebræ (but not of the intervertebral bodies) causes pain and tenderness in the back, and, if the spinal cord be reached, paraplegia will follow. Rupture into the œsophagus or into the mediastinum (causing diffuse aneurism) may occur.

Variations occur in the above three cases, according to the exact position of the sac. If the aneurisms bulge upwards, there may be pain in the shoulder and down the arm from pressure on the brachial plexus; and if the sympathetics in the neck be compressed, the pupil is at first dilated from irritation, and then contracted from paralysis of the radiating fibres of the iris. The second sound at the aortic valves is often accentuated.

Aneurism of the Abdominal Aorta.

This may occur at any part. As regards the aneurismal signs, care must be taken in observing that the pulsation is expansile, because (*a*) in

nervous persons, especially women, the aorta may pulsate very greatly; (*b*) solid tumours may have pulsation communicated to them by the aorta beneath; (*c*) large malignant tumours of the vertebræ may pulsate. But in none of these is the pulsation expansile. As regards the bruit, the artery must not be compressed by the stethoscope, because that alone may produce a bruit. If the bruit be heard in the back, it assures the diagnosis.

As regards pressure, the stomach may be displaced, causing dyspepsia. The solar plexus may be compressed, causing pain, syncope, and other sympathetic disturbances; the common bile-duct, causing jaundice; the renal veins, causing scanty, high-coloured, albuminous urine; the inferior vena cava, causing œdema of the legs, and perhaps great enlargement of the superficial abdominal veins (see p. 60), etc. Most often the vertebræ are eroded, causing pain and tenderness in the back, and perhaps later on paraplegia; and pressure on the spinal nerves may cause pain (and sometimes an eruption of herpes) in the course of the nerves.

Treatment.—Fibrin is often deposited on the inner surface of an aneurism in successive layers, forming the *laminated clot*. If this clot become organized to fibrous tissue by the growth of granulation tissue into it, the sac would be filled up by fibrous tissue. This would be a *spontaneous cure*, but it does not often occur. For aortic aneurisms (in which ligature and pressure are impossible) such measures as galvano-puncture, the introduction of wire, etc., into the sac, the scratching of the inner surface with needles, etc., have been tried with the object of causing coagulation and subsequent organization.

The rest of the treatment consists in rest, a moderate diet, and the administration of iodide of potash to lower the blood-pressure. Morphia must be given to relieve pain.

THROMBOSIS AND EMBOLISM.

Thrombosis means coagulation of blood in the heart or bloodvessels. The clot is called a thrombus. For thrombi in the heart, see Endocarditis. In vessels the coagulation is due to death of the endothelium, caused by injury, or acute inflammation of the wall, or chronic endarteritis, especially syphilitic, etc. Slowing of the blood-stream predisposes to thrombosis. A thrombus formed at one point may extend for a long distance along the vessel (*e.g.*, thrombosis in the uterine veins may extend to the common iliac). In arteries thrombosis often forms behind an embolus.

If the thrombus be aseptic, granulation tissue from the wall of the vessel grows into it, replaces the clot, and then becomes fibrous, so that the vessel is converted into a fibrous cord. Sometimes before this happens, a piece of the clot is detached and carried along the blood as an embolus.

Septic thrombi contain micrococci, and are soft and purulent-looking. They usually break up and distribute septic emboli, which cause pyæmic abscesses in other organs. Arterial thrombosis is most common in the brain, and most often occurs in syphilis or in the puerperal state. The symptoms depend on the artery which is blocked, but as the middle cerebral is most often affected, hemiplegia usually follows. The paralysis develops in the course of from half an hour or more, and there

may be very little cerebral disturbance (such as coma, etc.). True softening of the brain may follow.

Arterial thrombosis in the limbs may cause gangrene. Venous thrombosis is the more common, and follows phlebitis. It causes localized pain and œdema of the distal parts, and, if it can be felt, the vein is tender and cord-like. It is common in the *lateral sinus* of the skull as a consequence of necrosis of the temporal bone in ear disease. These cases are septic, and a great cause of general pyæmia and suppurating infarctions in the lungs.

The *portal vein* may be thrombosed (pylephlebitis), and lead to abscesses in the liver.

The *femoral vein* is often thrombosed—in typhoid fever, the puerperal state, etc. The œdema of the legs usually clears up (from dilatation of other veins), but is apt to recur on getting up.

In many septic cases affecting the limbs there is lymphangitis as well as phlebitis. Then the œdema is firmer and the limb whiter, and complete recovery more rare.

The nature of **Emboli** has already been explained (see p. 46). Emboli become arrested when they reach a vessel too small to let them pass. Those in the veins are arrested in the lungs, or, in the case of the portal vein, in the liver. Those from the left side of the heart or the arteries reach the limbs or various viscera.

If an embolus blocks a terminal artery (*i.e.*, an artery which has no anastomosis), the piece of tissue supplied by that artery dies, and is called an *infarction*. Infarctions are pyramidal in shape, and red if hæmorrhage occurs into the dead tissue, but otherwise white. They occur in the lungs,

spleen, kidney, brain, eye, etc.; but not in the liver, which has no terminal artery.

If the embolus be septic, an abscess forms round it, whether it has blocked an artery or only a capillary.

RAYNAUD'S DISEASE—SYMMETRICAL GANGRENE.

In its most extreme development this disease is characterized by gangrene of the fingers, toes, or ears. It always affects the two sides of the body symmetrically, and the affected parts become dry and black.

But the disease may only reach its milder stages. The first stage consists in numbness, coldness, and whiteness of the extremities, say the fingers. The pallor means anæmia, which is thought to be due to spasm of the arterioles. This stage has been called *local syncope*.

The stage of pallor may be succeeded by a stage of active hyperæmia, the fingers becoming bright red, hot, and rather swollen and painful.

In the third stage the fingers become blue, cold, and swollen—in fact, cyanosed from passive hyperæmia. This stage has been called *local asphyxia*, and it may come on without the previous pallor.

Finally, the gangrene may occur, but it may not be preceded by all the stages.

In some cases the blood contains free hæmoglobin, as in paroxysmal hæmoglobinuria. The disease may be hereditary, and in its milder stages it may occur in attacks which are usually excited by cold.

The **Treatment** consists in careful avoidance of cold, and in giving tonics.

DISEASES OF THE LYMPHATIC SYSTEM.

Inflammation of lymphatic vessels is called *lymphangitis*, and is a common result of septic infection of a wound. In superficial parts the lymphatics appear as red lines under the skin.

The dilatation of lymphatic vessels is called *lymphangectasis*, and is due to some obstruction of a lymphatic trunk. Thus, the *Filaria sanguinis hominis* may block the lymphatics of the urinary passages, and cause chyluria. The same parasite may block the main lymphatics of the thigh, in which case the legs become white and œdematous (the œdema being, however, more solid than the ordinary serous œdema of heart disease), and the skin and connective tissue become hypertrophied, so that the limb gets enormous, and the affection is called *elephantiasis*. If the thoracic duct or receptaculum chyli be obstructed, the lacteals of the mesentery become distended, and chylous ascites may follow. Signs of starvation are said to occur in these cases.

Enlargement of Lymphatic Glands—Causes.—Lymphadenitis, acute, subacute, or chronic; lymphadenoma; tuberculosis; cancer.

Pathological Anatomy.—In *acute lymphadenitis* the glands are large, soft, deep red, but sometimes dotted with yellow points, which may suppurate. They occur in the neighbourhood of acute inflam-

mations, *e.g.*, in the neck from scarlatinal or other severe sore throats, and in the mesentery from typhoid ulceration of the ileum. In diphtheria the cervical glands are firmer, and microscopically contain fibrin.

In *subacute lymphadenitis* the glands are like the acute, but the changes are less marked. In the neck they follow subacute sore throats, and they may occur in various situations.

In *chronic lymphadenitis* the glands are not so large, and appear fibrous. They occur in syphilis, and may follow on acute lymphadenitis.

In *lymphadenoma* the glands are large, just firm, and white. In severe cases, called lympho-sarcoma, they may grow so as to fuse; but they should not suppurate, and are not matted together by inflammatory adhesions. They are common in the neck and elsewhere.

In *tuberculosis*, in early cases, grayish-white nodules are seen in the glands. These caseate and become yellow, and, finally, the whole gland may be a cheesy mass, encapsuled by fibrous tissue. The glands may be matted together. But the tubercles may also suppurate or calcify. They are common in the neck (where they used to be called *scrofulous glands*), in the mesentery, etc.

In *cancer* there is a definite pinkish-white growth in the glands.

Clinically.—In *acute lymphadenitis* the glands enlarge rapidly, and are very tender and painful. If they suppurate, the skin over them becomes red; and if they are allowed to burst, puckered scarring may result. The subacute form presents the same symptoms in less intensity. In the chronic form the glands may feel stony.

In *lymphadenoma* the glands are painless (see below).

In *tuberculosis* the glands enlarge slowly, and often by fits and starts. They are sometimes painful and tender, but at other times not. They may feel as if matted together, and if left to burst produce great scarring. A family history of tuberculosis and the presence of tubercular lesions elsewhere may assist the diagnosis.

In *cancer* the glands enlarge slowly, and are usually tender. A primary growth can usually be found.

LYMPHADENOMA—HODGKIN'S DISEASE.

Pathology.—In this disease there is an increased growth of lymphatic gland tissue in the lymphatic glands and spleen, and the same lymphoid growths appear in organs like the liver and lungs, in which lymphoid tissue is not normally apparent. It must be considered a malignant disease like sarcoma.

For a description of the glands see above. Peyer's patches and the solitary glands (being lymphoid structures) may also be large, white, and firm. The *spleen* may be very large, and dotted all over with round white nodules (like marbles, but both larger and smaller). These are the enlarged Malpighian bodies. This spleen has been called the *hard-bake* spleen, from the presence of the white nodules on the dark background of spleen pulp. In some cases large white wedge-shaped masses are found in the spleen when the lymphoid growth has involved a whole lobe.

In the *liver*, etc., white tumours of various sizes may be found. The *heart* is often fatty.

Symptoms.—The *glands* become enlarged, but are not painful or tender. In the neck, axilla, and groin, they may be felt as lobulated masses, not adherent to the skin. They should not suppurate. The disease has been classified into cervical, thoracic, and abdominal types, according to whether the cervical, thoracic, or abdominal glands enlarge first and most extensively.

The *spleen* can usually be felt, and is not tender. It may be irregularly enlarged.

The general symptoms are those of progressive asthenia, the patient becoming weaker (but not thinner at first) and having a weak pulse, habitual dyspnœa, and, later, attacks of syncope. The number of red blood-corpuscles is diminished, and there is some increase of the white. The face is pale. There may be attacks of fever (temperature 103° or more) from time to time, which last a few days. The new growths may press on various parts, and give rise to symptoms. This is especially the case when the mediastinal glands are very large, for they may compress the trachea, œsophagus, or the veins, etc., causing dyspnœa, dysphagia, and œdema of the head, neck, and arms, etc. In the abdominal form the gland masses may be felt, the liver may be large, painless, and usually smooth, and diarrhœa may result from the affection of Peyer's patches.

Rarely the growths appear in unexpected places, such as within the vertebral canal, causing paraplegia by compression of the cord.

Under **Treatment** temporary improvement may occur, the glands becoming smaller. *Liquor arsenicalis* in large doses does most good.

DISEASES OF THE SPLEEN.

All recognised diseases of the spleen are associated with *enlargement* of the organ, and in many cases it is *painful* and *tender*. The *blood* may be altered, and, in particular, an excess of white corpuscles is often present.

Enlargement of the spleen is ascertained by *percussion* and *palpation*. The area of normal splenic dulness should extend vertically from the ninth rib in the mid-axillary line to the lower border of the eleventh rib. On either side of this vertical line dulness may be obtained for about $1\frac{1}{2}$ inches, the shape of the dull area being oval, with the long axis running from behind forwards and slightly downwards.

The spleen cannot be felt unless it be enlarged or pushed downwards. When felt, its somewhat pointed lower end and the notches on its anterior border are quite characteristic. It may be so large as to reach to the pelvic brim and beyond the umbilicus; but it always keeps in front of the descending colon, so that there is resonance in the loin behind it. The colon is always in front of tumours of the left kidney.

Causes of Enlargement of the Spleen.—Malaria, lymphadenoma, leucocythæmia, lardaceous disease, typhoid fever and other fevers, infarction, abscess, rickets, syphilis, tuberculosis, cancer or sarcoma, hydatids, cirrhosis of the liver.

Malaria.—The spleen may be enormously enlarged. At first it is soft, but later becomes fibrous, and therefore firmer (constituting the *ague cake*). It is often almost black from pigmentation. A malarial spleen may rupture from comparatively slight violence.

Lymphadenoma.—See p. 107.

Leucocythæmia.—In these cases the spleen may be very large, but not materially altered in other respects.

Lardaceous Disease.—The spleen is large, firm, pale, and dotted all over with small round nodules, which are gray and translucent, like grains of boiled sago. Hence the name *sago-spleen*. These nodules are the Malpighian bodies, converted into lardacein, and they stain dark brown with iodine. Microscopically (when stained with methyl violet, which stains lardacein lake-red and the normal tissues blue) the lardacein is first seen in the walls of the arterioles attached to the Malpighian bodies, and later on in the whole Malpighian body. In other cases there are large masses of lardacein in the spleen.

If there be a history of suppuration or syphilis, and if the liver or other organs be affected, the diagnosis is clear.

In **Typhoid** the spleen is large and soft. Clinically it can usually be felt at the end of the second week, and it is tender.

Infarctions in the spleen are nearly always non-hæmorrhagic (see p. 103). They form white firm wedge-shaped masses of tissue, but there is often a thin rim of hæmorrhage round them. At the time when the artery becomes blocked there is usually a sharp pain in the splenic area, and then

the enlargement can be felt. They occur in endocarditis, especially in the septic form. In the latter case they may suppurate, and lead to abscess.

Abscesses may occur in pyæmia, apart from infarction, but are rare.

In **Rickets** the enlargement of the spleen is moderate, and it is a simple hypertrophy. The same remark applies to **Syphilis**, except when syphilis causes lardaceous disease or gummata.

Tuberculosis.—The spleen may be thickly studded with miliary tubercles without being much enlarged. They may be minute and gray, or larger, yellow and caseous.

Cancer and Sarcoma are not common in the spleen. **Hydatids** occur occasionally, and may be recognised by the hyaline wall. The fluid in the cyst is non-albuminous, and of low specific gravity.

Cirrhosis.—The spleen gets large from congestion at the time that the liver begins to shrink.

In cases of cardiac back-pressure the spleen, although probably enlarged at an early stage, soon becomes small and fibrous.

DISEASES OF THE BLOOD.

Anæmia results from loss of blood, and from a defective supply of nourishment to the blood, such as occurs from starvation, disease of the nutritive organs, and diseases like cancer, which take the nourishment intended for the body. Ischæmia means a local anæmia of a part due to narrowing of the artery supplying it. But there are certain diseases in which an alteration in the blood is the most prominent change, and until we can find out the cause of them we must call them *idiopathic anæmias*. They may be due to structural disease of the organs which make the blood, or to poisons circulating in the blood (probably absorbed from the intestine) which act injuriously on its corpuscles or its chemical substances.

CLASSIFICATION OF ANÆMIAS.

1. Simple anæmias, in which the constituents of the blood are simply altered in quantity. These are subdivided into :

(a) Chlorosis, in which the hæmoglobin is diminished.

(b) Oligocythæmia, in which the red blood-corpuscles are fewer.

(c) Leucocytosis, in which the white corpuscles are simply more numerous.

These three types may be present together, but one or other may predominate in any particular case. Hyp-albuminosis means that the blood is deficient in albumen.

2. Malignant anæmias, in which there are not only quantitative but qualitative changes in the blood. These usually end fatally. They are subdivided into — (a) Pernicious anæmia, (b) leucocythæmia.

In the anæmia from hæmorrhage, starvation, etc., there is marked oligocythæmia. In malignant diseases and in debility from any cause there may be leucocytosis. The normal proportion of white to red blood-discs is about 1 to 500.

In pregnancy there is a true hydræmia, the corpuscles being apparently diminished because the fluid is in excess.

The common anæmia of young women is called Chlorosis, because that is the chief change in the blood.

Causes.—It has been attributed to the absorption from the intestine into the blood of poisons derived from retained fæces, the theory being based on the frequency of constipation in these cases, and their curability by purgatives. Other views are that the development of the body (and of the sexual organs at puberty in particular) throws a strain on the nutritive qualities of the blood, which the organs of nutrition cannot keep pace with. Many cases occur in young women who work hard at sedentary occupations, and who do not get sufficient fresh air, sunlight, and exercise.

Symptoms.—There is greenish-yellow pallor of the face, conjunctivæ, lips, and buccal mucous membrane. There is dyspnœa and palpitation on

exertion, and sometimes attacks of syncope. There is a thrill, and a continuous humming murmur called the *bruit de diable*, over the veins of the neck, and often a systolic bruit over the arteries. A systolic bruit over the pulmonary valve is common, and there is often a systolic bruit and thrill at the apex; but the pulse, although weak, is not small, as in mitral disease. The above cervical and cardiac murmurs may occur in all forms of anæmia. Edema of the feet is common.

Indigestion is usually complained of, and in particular a burning pain in the stomach immediately after food. This pain may lead to vomiting. Constipation is almost always present. The urine is pale and abundant, and there may be amenorrhœa for months. The patient is often fairly fat.

The blood shows a marked deficiency of hæmoglobin, and some oligocythæmia.

Treatment.—Iron must be given to increase the hæmoglobin. *Ferrum redactum* is the best form, but the carbonate of iron is also good. Constipation should be treated with aloes, or magnesium sulphate, or habitual enemata. Fresh air and moderate exercise are essential.

Pernicious Anæmia.—This is most common in middle-aged men. There is not merely an extreme amount of oligocythæmia (the red discs sometimes falling below 20 per cent.), but the red blood-corpuscles are, some of them, smaller than normal (microcytes), others larger than normal (macrocytes), others abnormal in shape (poikilocytes), and they do not form rouleaux. In addition, large nucleated red blood-corpuscles (called megalocytes) may be found in the worst cases. The individual corpuscles may contain an excess of hæmoglobin, and the blood

contains free hæmoglobin. There may be some leucocytosis. The other symptoms are like those of ordinary anæmia—that is, there is debility, dyspnœa, bruits, etc. But in addition attacks of vomiting occur from time to time; and hæmorrhages may take place in the retina, from the mucous membranes, under the skin, etc. The urine is high-coloured from excess of urinary pigments, and the spleen may be enlarged. There may be no emaciation. Finally, the heart gets weaker and weaker, and serious attacks of syncope occur, in one of which the patient dies.

After death the fat has a peculiar lemon colour. The heart shows fatty degeneration, visible to the naked eye as a yellowish striation or dotting of the myocardium, particularly in the muscoli papillares. The liver is fatty, and the nuclei of the liver-cells may be very large. Also free iron has been found at the periphery of the liver lobules, and is supposed to be deposited there from the free hæmoglobin which comes to the liver in excess by the portal vein. The spleen may be hypertrophied, and show the varieties of corpuscles seen in the blood during life. The marrow of bones may show the same corpuscles with excess of marrow cells, and to the naked eye it is pink and diffuent rather than fatty. Lastly, submucous and subserous hæmorrhages may be found.

Leucocythæmia.—In this affection the leucocytes in the blood are not merely in great excess (perhaps there may be one white to twenty red), but many of them are very large, and contain large nuclei. They may also contain particles which can be stained with eosine (eosinophil corpuscles). Elongated octahedral crystals (Charcot's crystals) may

also be found in the blood. The other symptoms are those of severe anæmia, the pallor being very marked, and the dyspnœa, faintness, etc., being as bad as in pernicious anæmia. Hæmorrhages also may occur. The spleen is usually enlarged, and not painful. In other cases the lymphatic glands are large. From time to time during the affection there may be high fever for a few days. The prognosis is exceedingly bad.

Post-mortem.—The capillaries of all the organs are crowded with leucocytes. This is particularly marked in the liver, where the capillaries are in places so dilated as to compress the liver-cells. Collections of leucocytes may also be seen outside the capillaries. The heart may be fatty. The spleen is enlarged, and the kind of leucocytes seen in the blood during life may be found in it. If the case lasts a long time, the fibrous stroma of the spleen becomes increased in quantity.

Treatment.—Both pernicious anæmia and leucocythæmia should be treated with large doses of arsenic, under which temporary, or sometimes apparently permanent, benefit may occur. Preparations of bone-marrow are being tried.

OTHER BLOOD ABNORMALITIES.

In gout and other uric acid affections there is an excess of uric acid in the blood (lithæmia) in the form of urates of the alkaline metals.

In rheumatic fever there is said to be an excess of lactic acid in the blood.

In malaria the protozoon of Laveran is found in the blood, and in some stages of its development it is pigmented, and as the leucocytes, and perhaps

also the endothelial cells, are pigmented, the condition is called melanæmia—that is, black or pigmented blood.

Among other parasites found in the blood the embryo *filariæ* must be remembered, and as regards bacteria, anthrax bacilli and the spirilla of relapsing fever can usually be found in the blood during the diseases they cause.

HÆMATOPHILIA.

Patients affected with this disease are called *bleeders*, or are said to have the *hæmorrhagic diathesis*, because they bleed profusely (and even fatally) after very slight lesions, such as a scratch or slight cut, the extraction of a tooth, etc. Hæmorrhages from the nose or other mucous membranes, or into serous cavities or joints, or under the skin, may also occur. In some cases there is an inflammation of the joints, the nature of which is obscure.

The disease is congenital and almost confined to men, and it is strongly hereditary in the following way. The male and female children of a man affected with this disease are not likely to have it. And his son's children escape. But the sons of his daughters are very likely to have it. The pathology is obscure. Some think that the walls of the capillaries are abnormally thin. In many cases the blood takes longer to coagulate than normal.

Treatment.—Turpentine, ergot, and other vaso-constrictors, may be tried, and cold or a considerable degree of heat may be applied locally. Chloride of calcium in 5 grain doses may cause the blood to coagulate, and so stop the bleeding.

DISEASES OF THE THYROID GLAND.

MYXŒDEMA.

This disease is due to atrophy of the thyroid gland, and follows complete removal of the gland by operation. It is most common in middle-aged women, but may be found in idiot children. It is supposed that the blood, as it passes through the thyroid, absorbs some material from the thyroid juice which is essential to the nutrition of the tissues, and that when this material is absent from the blood (as it must be after removal of the thyroid), the changes which cause myxœdema follow.

After death the connective tissue of the skin and other parts is found to be infiltrated with a fluid containing (in recent cases) much mucin. In old cases the connective tissue may be increased in quantity.

Symptoms.—The patient becomes slow in moving and in thinking. She speaks slowly, and answers a question after a considerable interval, but correctly. In old cases the intellectual condition may pass on to complete imbecility. The face is apathetic and waxy-looking, with a pink coloration on the cheeks. The eyelids, edges of the nose, and the lips, are thickened, pale, and œde-

matous-looking. The hair is thin and dry. Perspiration is slight, and the urine is scanty. The arms show the thickening of the skin well, and the hands are large and flat, and have been compared to spades. The legs look œdematous, but do not pit readily on pressure. The patient feels cold, and there is usually amenorrhœa.

Treatment.—All the symptoms can be removed, or their occurrence prevented by continuously feeding the patient on the thyroid glands of the sheep, minced and lightly cooked. Or preparations containing thyroid juice can be swallowed or injected hypodermically. But on stopping the treatment the symptoms recur. Iron and other tonics may be given.

BRONCHOCELE—GOITRE.

In this disease the thyroid gland becomes enlarged by the distension of its acini with an excess of thyroid juice. In this way cysts are formed which contain a more or less colloid material. The shape of the gland is altered according to the situation of the cysts on one side or the other, or on both, and it feels more or less fluctuant. There is no pain. If the size of the gland be great, it may produce symptoms by compressing the trachea or œsophagus, etc., but otherwise no other symptoms arise.

The disease is common in Switzerland and in Derbyshire, and it has been attributed to drinking water impregnated with magnesian limestone. It is common in cretins.

Treatment.—Preparations of iodine should be given internally, and iodine may be painted on

over the thyroid or injected into the larger cysts. The whole of the gland should never be excised, because myxœdema would follow.

GRAVES' DISEASE—EXOPHTHALMIC GOITRE.

There are three characteristic symptoms of this disease, namely, enlargement of the thyroid, palpitation of the heart, and exophthalmos, *i.e.*, protrusion of the eyeball. The last is also called proptosis.

The enlargement of the thyroid may be cystic, as in the common goitre, or produced by great increase in the connective tissue, or by great dilatation of the vessels. In the last case the gland may pulsate.

The proptosis is due to dilatation of the vessels and increase of the connective tissue and fat of the orbit outside the eyeball. Some think it is due to muscular action.

The dilatation of the vessels of the thyroid and of the orbit, and the palpitation of the heart, suggest that the disease is due to a disturbance of the sympathetic ganglia of the neck. But no lesion can be found in those ganglia, so, if sympathetic, the disturbance must be functional. Others think that the disturbance may take its origin in the medulla oblongata.

The latest view is that the symptoms are due to the excessive absorption of thyroid juice from the enlarged thyroid by the blood. This theory is based on the fact that the symptoms of this disease are almost the opposite of those of myxœdema, and as myxœdema is due to the absence of thyroid juice, Graves' disease should be due to an excess of it. Further, if thyroid juice be given in excess

for myxœdema, such symptoms of Graves' disease as flushings, palpitation, etc., may result.

Symptoms.—The enlargement of the thyroid is moderate, and usually fairly symmetrical. As has been said, it may pulsate, and a bruit may be heard over it. The exophthalmos may be recognised at an early stage by the fact that, if the patient is made to look downwards suddenly, the eyeball comes down, but is not followed by the eyelid. Later on the eyeball is glaringly prominent.

The palpitation is very distressing to the patient, and may lead to hypertrophy of the left ventricle, and later on to dilatation, so that even valvular incompetence may occur. There is a tendency to attacks of flushing of the face, with sweating, and the patient usually has an excited appearance.

If the disease occur in young women, anæmia is usually present, and there may be various symptoms of sympathetic disturbance.

The prognosis is always serious, but some cases recover in a remarkable way.

Treatment.—Iron and other tonics, belladonna, ergot, and digitalis, to constrict the vessels, and various other remedies, have been tried. Weak continued electrical currents from the back of the neck to the thyroid have been recommended. Excision of a large part of the thyroid has been tried, from the point of view that the symptoms are due to excessive absorption of thyroid juice from the gland, and good results are claimed for this treatment.

ADDISON'S DISEASE.

Pathology.—The disease is nearly always due to the growth of tubercles in both suprarenals. The tubercles caseate and convert the whole organ into a fibro-caseous or cretaceous mass, surrounded by fibrous tissue, in which the semilunar ganglia and solar plexus may be involved. In a few cases the suprarenals are merely atrophied. The pigment granules are found in the deep cells of the epidermis.

Symptoms.—There are three groups of symptoms: (1) progressive asthenia, (2) pigmentation, and (3) gastric crises.

1. The patient becomes steadily weaker, gets dyspnœa on exertion, and has attacks of syncope and giddiness. Towards the end he may not be able to move without fainting. There may be no emaciation.

2. The pigmentation produces a peculiar bronzed appearance of the skin. It occurs in three kinds of places: (*a*) where pigment exists naturally, such as the nipple, axillæ, etc.; (*b*) on exposed parts, such as the face and hands; (*c*) on parts that are irritated by pressure, friction, etc., such as the shoulders where rubbed by the braces, on the seat of a blister, around a recent wound, etc. An old white scar does not become pigmented. Pigmenta-

tion may also be seen in the mouth, where the teeth are apt to nip the mucous membrane. The pigmented areas shade off gradually at their edges. Death may occur from asthenia before there is any pigmentation.

3. Gastric crises are *attacks* of epigastric pain and vomiting, which occur without any obvious cause, and are believed to be of nervous origin. They may occur at any time during Addison's disease, and are probably due to irritation of the solar plexus.

Differential Diagnosis.—Addison's disease resembles *pernicious anæmia*, as in both there are progressive asthenia and attacks of vomiting. The differences are the condition of the blood in the anæmia (particularly megalocytes) and the pigmentation in Addison's disease.

The coloration in *jaundice* can be distinguished from the bronzing of Addison's disease by the fact that the conjunctivæ and urine are bile stained. In a pigmentary disease called *leucoderma* there are brown patches around the bleached patches of the skin, but there is a sharp line of demarcation between the pigmented and the white patches, while in Addison's disease the brown patches shade off gradually.

Treatment.—We can only enjoin rest, warmth, and good food, and prescribe tonics.

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AIDS TO MEDICINE.

PART III.

DISEASES OF THE LIVER, PANCREAS, ALIMENTARY
CANAL, PERITONEUM, AND KIDNEY.

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PREFACE.

ANY attempt to include the essentials of a large subject within the compass of a small book necessitates the omission of many facts which seem important to the writer, and will doubtless be missed by the reader. Nevertheless, it is hoped that there is enough matter arranged in a convenient form in this book to make it a real 'Aid' to all those who are studying medicine.

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4, MANSFIELD STREET,
CAVENDISH SQUARE, W.
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AIDS TO MEDICINE.

DISEASES OF THE LIVER.

IN all cases of hepatic disease, it is necessary to make a *physical examination* of the liver; to note the presence or absence of *pain and tenderness*, and of *jaundice*; and to look for symptoms of *obstruction* of the *portal vein*, or *inferior vena cava*. In rare cases cerebral symptoms, due to what is called *bilious toxæmia*, may occur, and hæmorrhages, other than those due to portal obstruction, may be met with. It is, of course, necessary to investigate the condition of the alimentary canal, urine, etc., and the *subjective symptoms* complained of by the patient may be of great importance.

PHYSICAL EXAMINATION OF THE LIVER.

Percussion.—The upper limit of liver dulness extends from the base of the ensiform cartilage, through the fifth intercostal space in the right nipple line and the seventh interspace in the axillary line, to the eleventh interspace at the back. All along and above this line there is a zone, about half an inch high, of partial liver dulness, where the liver is overlapped by the lung. The lower limit of dulness follows the lower edge of the ribs in the right hypochondrium; while, in the epigastrium, if the stomach be empty, an inch or two of liver

dulness may be obtained. A dilated colon or stomach interferes much with the lower limit of liver dulness, even when the liver projects below the ribs. Hence both deep and superficial percussion should be used.

Palpation.—If the liver be enlarged and project below the ribs, it can be felt, and it is necessary to note whether it be hard or soft, smooth or uneven. If, however, the enlargement be slight, a fat abdominal wall or a rigid rectus may obscure these points. By palpation and percussion it can be ascertained whether the liver be small or enlarged.

Enlarged livers are divided into those which are *smooth*, and those which are *uneven*. The division into painless and painful enlargements is less satisfactory.

The *large smooth livers* are caused by :

Acute congestion	Moderately painful.
Mechanical congestion	Painful from time to time.
Fatty disease	Painless.
Lardaceous disease	Painless.
Retention of bile	Usually painless.
Cirrhosis in early stage	Usually painless.
Miliary tuberculosis	Painless.

The *large uneven livers* are caused by :

Abscesses of all kinds	Usually very painful.
Cancer of all kinds	Usually painful.
Hypertrophic cirrhosis (late stage)	Painless.
Old gummata	Painful from time to time.
Hydatids	Painless.

The *small livers* are caused by: Atrophic cirrhosis (late stage); red atrophy; and acute yellow atrophy.

Enlargement of the liver may be simulated by displacement of the organ (*e.g.*, from the pressure of stays or by tumours), by abdominal tumours, or by conditions causing dulness at the base of the right lung. In pleuritic effusion the upper line of dulness is straight, while that of the liver is curved.

At a post-mortem the liver should weigh about 50 oz., and be quite smooth.

Pain and Tenderness.—It is important to distinguish constant pain from the pain which is only produced by pressure or rubbing. For instance, a nodule of cancer in the liver may cause no pain until it be touched. Again, livers may be painful at one period of a disease and not at another; for instance, in mechanical congestion there is pain while the liver is being distended by blood, but not after the distension has become permanently established. A great deal of pain in liver disease is due to inflammation of the peritoneum over the organ (perihepatitis). A large liver may cause pain by dragging on its attachments, particularly if the patient lies on the left side.

Jaundice, or Icterus.—In jaundice the blood contains bile, which exudes from it and stains all the fluids and tissues of the body. The *symptoms* of jaundice are: the yellow colour of the skin and conjunctivæ; bile in the urine (for tests, see Diseases of the Kidney); the absence of bile from the stools, which become pale and hard like clay, and very offensive, and may contain fat; a slow pulse and low temperature (unless the jaundice complicates a fever or inflammation); itching of the skin in old standing cases; yellow vision some-

times ; mental depression ; a disgust for food ; and dyspepsia.

The *causes of jaundice* are divided into *obstructive* and *non-obstructive*. In obstructive jaundice there is some obstruction of the common bile-duct or of the small ducts in the liver, so that the bile cannot escape into the intestine. The lymphatics in the wall of the ducts then absorb the bile, which eventually reaches the blood by way of the thoracic duct, and circulates in the bloodvessels. In non-obstructive jaundice no obstruction to the escape of bile from the ducts can be found, and the occurrence of jaundice in these cases is explained as follows. It is known that the greater part of the bile which reaches the intestine is normally re-absorbed by the bloodvessels of the bowel, and that as soon as it reaches the blood it is converted into blood pigment and colourless materials. Now, if the chemical changes going on in the blood be abnormal, as in certain cases of poisoning or of fever, then the reabsorbed bile will not be altered, but will remain as bile, and produce the symptoms of jaundice.

Other authorities think that in non-obstructive jaundice there is a true suppression of bile, *i.e.*, that bile is formed in the blood in the liver, and is not removed from the blood by the liver-cells.

It has been shown, of late, that certain cases of jaundice, formerly supposed to be non-obstructive, are due to obstruction of the minute ducts in the liver.

Obstruction of the common bile-duct may be recognised by the complete absence of bile from the stools, and by, in most cases, a palpable enlargement of the gall-bladder. If of long duration, the jaundice gets very dark.

In obstruction of the small ducts in the liver, some bile will be present in the stools (because all the ducts are not blocked); the gall-bladder will not be distended, and the jaundice does not become very dark.

It is said that obstructive jaundice of either kind can be distinguished from non-obstructive jaundice by the fact that in the former the urine contains both bile pigment and bile acids, while in non-obstructive jaundice it contains bile pigment only.

The Causes of Obstruction of the Common Bile-duct are: 1. Causes *inside the duct*—Gallstones, mucus (produced by catarrh), and worms. 2. Causes *in the wall of the duct*—Swelling of the mucous membrane from catarrh; fibrous stricture from cicatrization of an ulcer of the duct or of the duodenum; cancer of the duct; congenital obliteration of the duct. 3. Causes which *press on the duct from outside*—Cancer of the liver, or of the adjacent glands or pancreas; aneurism, especially of the hepatic artery.

The Causes of Obstruction of the Small Ducts in the Liver are: Cirrhosis; congestion and œdema of the ducts (as in the nutmeg liver); poisoning by toluylendiamine.

The Causes of Non-Obstructive Jaundice are: Poisons like phosphorus or antimony; the poison of snake-bite; the poison of fevers, like yellow fever, relapsing fever, remittent malarial fever, croupous pneumonia, etc.; acute yellow atrophy of the liver; mental emotion.

The treatment of jaundice is that of the cause. It is said that, after the cause has been cured, the disappearance of the yellow colour from the skin can be hastened by giving benzoate of soda

(grs. xv. t. d.) or dilute nitro-hydrochloric acid (internally or as a bath). The itching of chronic jaundice may be relieved by hot baths, or by a hypodermic injection of nitrate of pilocarpine (gr. $\frac{1}{8}$).

Portal Obstruction.—The main portal vein may be compressed by a cancerous nodule, etc.; or the portal branches inside the liver may be constricted, as in cirrhosis. The consequences of portal obstruction are: Congestion and catarrh of the *stomach*, leading to *dyspepsia* and *hæmatemesis*; the same conditions of the *intestines*, leading to *melæna* and *hæmorrhoids*; congestion of the spleen, leading to enlargement; and, lastly, *ascites*. The symptoms of portal obstruction are often inversely proportional one to the other. For instance, the spleen may become much smaller after a profuse hæmatemesis.

Enlargement of the superficial abdominal veins occurs in many liver affections, because the inferior vena cava may be compressed by the liver or by the weight of ascitic fluid.

Cerebral Symptoms, such as headache and confusion of ideas, going on to delirium and coma, may occur in liver affections, especially in acute yellow atrophy and long-standing cirrhosis. With these symptoms there is usually tremor of the hands and a dry, brown, tremulous tongue; in fact, the condition is that of the *typhoid state*. If jaundice be present, the symptoms are called *bilious toxæmia*, and they are due to a poison circulating in the blood. The poison is not bile, but some substance which is produced by the incapacity of the liver to perform its normal functions.

Hæmorrhages, other than those due to portal

obstruction, such as epistaxis and subcutaneous petechiæ, may occur in liver diseases, especially in acute yellow atrophy, long-standing cirrhosis, and cancer. They appear to be due to degeneration of the capillary walls.

Subjective Sensations, such as giddiness, specks before the eyes, migraine, etc., may be due to functional affections of the liver.

THE FATTY LIVER.

Pathology.—Fatty degeneration of the liver—*i.e.*, the conversion of the liver-cells into oil—occurs in the fevers, in phosphorus-poisoning, etc. The same affection occurs, as a secondary change, in cirrhosis. But the large fatty liver met with in practice is due to an infiltration of the liver cells with oil. *Macroscopically*, such a liver is large, smooth, pale, and has a blunted edge, and on section it is greasy, and tears easily. *Microscopically*, oil globules are seen in the liver-cells, first at the periphery of the lobules.

The **Causes** of a large fatty liver are over-eating (particularly starchy foods), together with deficient exercise. Alcoholic excess may also cause it, and it is often found in phthisis.

The **Symptoms** of a fatty liver are dyspepsia, and a sense of weight and fulness in the right hypochondrium. On examination the liver is large and smooth; it feels soft, as a rule, and is not tender. There is no jaundice and no symptoms of portal obstruction, except, perhaps, piles. More especially the spleen is not enlarged. The urine should be normal.

Treatment.—Regulate the diet, insist on exercise,

and keep the bowels acting rather freely. Rhubarb, bitters like quassia and calumba, and carminatives like aq. menth. pip. or aq. anethi, are useful in treating the accompanying dyspepsia.

THE LARDACEOUS LIVER.

Causes. — Prolonged suppuration and syphilis. Those cases of suppuration which last longest, such as tubercular disease of bones, phthisis, chronic pyelitis, etc., are most likely to cause lardaceous disease.

Pathology. — Lardaceous disease of the liver (as of other organs) consists in the infiltration of the liver with a substance called *lardacein*. This substance is chemically an albuminoid, and is most closely allied to *fibrin*. It stains mahogany-brown with iodine, and lake-red with methyl-violet. *Macroscopically*, a lardaceous liver is large in all its dimensions, smooth, pale and firm, and its edge is blunted. On section it may be glistening. *Microscopically*, the lardacein is first seen in the walls of the *capillaries derived from the hepatic artery*. These lie in the intermediate zone of the liver lobule. Later on the amount of lardacein increases, and compresses the liver-cells.

Symptoms. — There is weight in the right hypochondrium, and the liver is enlarged in all parts, smooth, very firm, and not tender. There is no jaundice and no portal obstruction; but if the glands in the portal fissure become lardaceous they may cause pressure, and lead to both jaundice and ascites. The spleen is enlarged, not from portal obstruction, but from being itself lardaceous. Further, a lardaceous kidney may cause albuminuria,

and a lardaceous intestine diarrhœa. Finally, the presence of suppuration, or of syphilis, will complete the diagnosis.

Treatment.—Try to stop the suppuration, and then give tonics.

ACUTE CONGESTION OF THE LIVER.

This is supposed to be the pathological condition during a *bilious attack*, such as is induced by cold or by a surfeit of eating and drinking.

Symptoms.—Anorexia, nausea and vomiting, with a foul, dry tongue and thirst. These symptoms are, more probably, due to the accompanying active congestion of the stomach. There may be slight fever, headache, and giddiness. Later on there may be diarrhœa.

Physical Signs.—The liver is a little large and a little tender. There may be a slight degree of jaundice.

Treatment.—It is usual to give 5 grains of pil. hydrarg., and follow this with the well-known mist. alba (magn. sulph. and magn. carb.). Bismuth is also useful, and milk diet should be maintained for twenty-four hours.

It must be remembered that a local active congestion must always precede the formation of an abscess.

MECHANICAL CONGESTION OF THE LIVER.

Causes.—Obstruction to the escape of blood from the hepatic veins into the inferior vena cava and right auricle. Hence in practice it occurs in mitral regurgitation, mitral stenosis, and, more rarely, chronic bronchitis and emphysema.

Pathological Anatomy.—The liver is enlarged (particularly the right lobe), and smooth. On section, it presents the appearance of innumerable dark spots, each surrounded by a yellow ring. The dark spot is the distended hepatic veinlet in the centre of the lobule, and the yellow ring is formed by the liver-cells, which are fatty, and perhaps bile-stained. This black and yellow mottling has given the name of *nutmeg liver* to this condition. It tears easily.

Symptoms.—Those of the heart or lung disease; but there is a feeling of distension and weight in the right hypochondrium, and at times actual pain.

Physical Signs.—The liver is large, smooth, and may feel tense. It may be tender. Jaundice may occur from the blocking of the bile-ducts by congestion, and œdema of their walls. The spleen is too small to be felt, but ascites or hæmatemesis may occur if the congestion extends back from the hepatic to the portal vein. The urine is high-coloured, scanty, and albuminous from independent congestion of the kidney.

Treatment.—Treat the heart or lung condition; but particularly give watery purgatives (*e.g.*, pulv. jalap co. $\mathfrak{z}\text{i}$., quâque mane; or pulv. scammon. co., grs. xx., etc.) to empty the portal system, and so relieve the hepatic vein.

In very old cases of *nutmeg liver* fibrous tissue grows around the dilated veins, and by the subsequent contraction of this fibrous tissue the liver becomes small, and finely granular, but remains dark-red. This is called **Red Atrophy of the Liver**.

ACUTE HEPATITIS—ABSCESS OF THE LIVER.

The only form of acute inflammation of the liver which is of practical importance is that which leads to abscess.

Varieties of Abscess of the Liver.

1. **The Single, Large, so-called Tropical Abscess.**—This variety is, most often, a sequela of dysentery, and the *Amœba coli* (which is the parasite which causes dysentery) has been found in the contents of the abscess. It has also been attributed to malaria, tropical heat, and stimulating ingesta. The fluid inside the abscess is usually thick and chocolate-coloured, and not like ordinary pus.

2. **Small Multiple Pyæmic Abscesses.**—These are usually caused by the passage of septic micrococci from an ulcer of the intestine, along the mesenteric and portal veins, to the liver. The cocci become impacted in various capillaries of the liver, and the site of each impaction becomes an abscess, containing ordinary pus. Sometimes a septic thrombus forms in the portal vein itself, and then *pylephlebitis* is said to be present. The intestinal ulcers may be due to dysentery, ulcerative colitis, etc. These abscesses less commonly occur in the course of general pyæmia, due to wounds, etc.

3. **Abscesses due to Suppuration in the Bile-ducts.**—This condition has been called suppurative *cholangitis*, and it is due to a calculus which has become impacted in one of the large bile-ducts, and has set up inflammation and ulceration. The pus cannot escape because of the stone, and it therefore distends the ducts above the obstruction. The

abscess cavities in these cases are cylindrical, with smooth walls, and the pus is bile-stained.

4. Abscesses due to Suppuration of a Hydatid Cyst.

5. Abscesses due to Actinomycosis. — Actinomyces are a species of fungus which cause large tumours in cattle, and very rarely are found in the lungs and liver, etc., of man. An abscess due to this cause has peculiarly crumbling walls, and in the pus are small white particles visible to the naked eye, and composed of masses of the fungus.

6. Abscesses due to Suppuration of Tubercles or Gummata. — These are very rare.

Symptoms and Physical Signs. — These are: Remittent fever, sometimes moderate in degree, sometimes severe; severe rigors; profuse sweatings; emaciation; pain in the right hypochondrium and shoulder, sometimes very acute, but in a few cases absent; tenderness over the liver; rigidity of the rectus abdominis; sometimes œdema of the chest-wall; and sometimes actual pointing of the abscess. The tropical abscess may be recognised as a smooth, fluctuating projection from the liver; but if deeply situated, there will only be increased liver dulness. The small abscesses may be felt as nodules on the surface of the liver, but are usually not large enough to give fluctuation. Jaundice and portal obstruction do not occur in abscess of the liver, unless a large bile-duct or the portal vein be pressed on, which, on the whole, is not common.

Terminations. — The abscess may possibly become quiescent, the pus being encapsuled by fibrous tissue; but more commonly, if left to itself, it bursts either externally or into the peritoneum, pleura, stomach, etc. If the rupture be not imme-

diately fatal from peritonitis, etc., the abscess may be cured, or the pus may collect again.

Treatment.—The large abscess should be opened freely and drained. Multiple pyæmic abscesses can scarcely be treated in this way, and are, practically, always fatal.

PERIHEPATITIS.

This is an inflammation of the peritoneum over the liver, and of the subjacent Glisson's capsule. It occurs (1) as a part of general peritonitis, either acute or chronic (*e.g.*, tubercular); (2) as an extension of pleurisy through the diaphragm; (3) over an abscess, or cancer, or gumma in the liver. In acute cases fibrin or pus is seen on the surface of the liver; in chronic cases there are numerous fibrous adhesions between the liver and the diaphragm, etc. This last is best seen in syphilitic livers. Pain and tenderness in many liver affections indicate perihepatitis, but it must not be considered an independent disease. It can be treated by poultices, fomentations, or leeches, and later on by painting with iodine.

CHRONIC INTERSTITIAL HEPATITIS— CIRRHOSIS OF THE LIVER.

Causes.—The commonest of all is alcohol, in the form of spirits; but syphilis, and perhaps malaria, may also be causes. In some cases no cause can be traced. There is a form called *biliary cirrhosis*, in which the cause may be some irritating quality in the bile.

Pathological Anatomy.—The disease consists in an overgrowth of fibrous tissue between the lobules

of the liver. First of all granulation tissue (consisting of young connective-tissue corpuscles and new capillaries) is seen between the lobules, and later this develops into perfect fibrous tissue. Then the new fibrous tissue contracts and compresses the liver-cells, which atrophy or become fatty. In *biliary cirrhosis* abundant minute bile-ducts are seen in the new fibrous tissue. *Macroscopically* a typical cirrhotic liver is small, yellowish-white, very hard, and the surface may show nodules as large as *hobnails*, or may be finely granular. In some cases the liver is large, in spite of the cirrhosis. These have been called *hypertrophic* cirrheses, and the hardness and granular surface are then the characteristic points. These large cirrhotic livers occur in biliary and in syphilitic cirrhosis, because the granulation tissue develops between each lobule (unilobular cirrhosis), or even between the cells of the lobule (intercellular cirrhosis). The quantity of new tissue produced in the liver is thus very large, making the organ very big, and even when it becomes fibrous and shrinks, the size may remain larger than normal. In alcoholic cirrhosis, on the other hand, the fibrous tissue mostly grows around groups of lobules (multilobular cirrhosis), and is, therefore, not so great in quantity, and when shrinkage occurs, the liver gets quite small. Each *hobnail* is a group of lobules.

Symptoms and Physical Signs.—Cirrhosis is insidious in its onset, and variable in its manifestations, so that any of the following symptoms may be early or late in occurring, or may even be absent.

Sometimes *gastric symptoms*, such as loathing of food and morning sickness, are the first symptoms.

Sometimes the indications of *portal obstruction*

occur first. Thus, a profuse hæmatemesis, or *melæna* may be quite early; or the patient may come complaining of *piles*, or of abdominal enlargement due to *ascites*. The cause of the portal obstruction is the compression of the portal branches, between the lobules of the liver, by the contracting fibrous tissue. The *spleen* gets larger as the liver contracts.

At an early stage, and in *hypertrophic* cases, the liver may be felt to be large and hard. In *most* cases liver dulness is diminished. The abdominal veins may be prominent. *Jaundice* is rare, except in the biliary form, but the skin has often a brownish pallor. The patient may be thin or fat. Lastly, *epistaxis* and other hæmorrhages may occur, and the patient may become delirious, with a dry, brown tremulous tongue and other symptoms of the typhoid state, brought on by *toxæmia*.

Terminations.—With care the patient may live for years, but cannot be cured. Death may be due to hæmatemesis or epistaxis; to exhaustion from the loss of albumen in the ascitic fluid; to septic peritonitis after careless tapping; to *toxæmia*.

Treatment.—Only the symptoms can be treated. For the nausea give bismuth; or acid. hydrocyan. dil. with an effervescing mixture. For the appetite give *nux vomica* or other bitters with carminatives. Portal obstruction requires saline aperients, and the tapping of ascites. For profuse hæmatemesis, gallic acid, 10 grains, with opium, and for other hæmorrhages ergot or ol. terebinth., may be tried. Epistaxis may require plugging of the nares. In an early case alcohol must be stopped, and as regards diet the patient's taste is usually the best guide.

ACUTE YELLOW ATROPHY OF THE LIVER.

Causes.—The real cause is unknown. Some think that it is a general disease like the specific fevers. It is more common in hot climates, and the persons most liable to be affected are young adults, especially pregnant women and those who live freely.

Pathological Anatomy.—The liver is decreased in size and its capsule is wrinkled. On section it is soft and yellowish in colour, with, perhaps, some pink mottling. *Microscopically*, the lobular arrangement cannot be made out, and the liver-cells are degenerated, some being fatty, some granular, some pigmented, some swollen and cloudy, and some broken up altogether. But nearly all have lost their nuclei. In the pink parts groups of leucocytes may be seen, indicating an attempt at inflammatory reaction.

Symptoms.—The disease is not usually suspected until the grave symptoms occur. At the first, headache, with gastric symptoms, such as epigastric uneasiness and tenderness, with vomiting, may be present. But the prominent symptoms are those of the *typhoid state*, such as a dry, brown, tremulous tongue, subsultus tendinum, delirium, coma, or even convulsions. There may be fever, but the *temperature* is often *subnormal*, while the pulse is quick and feeble. *Jaundice* is present, but not intense, and the stools are not clay-coloured. Some say that this is because they contain not bile, but blood. The *urine* contains no urea, but leucin and tyrosin. It also contains bile pigment, and sometimes albumen and blood. *Hæmorrhages* are of frequent occurrence, and may be from the nose,

stomach, or any part of the body. Lastly, the shrinkage of the liver may be detected by percussion. The disease is rapidly fatal, and no particular treatment can be recommended.

HYDATID CYST.

A hydatid is the cystic stage in the life of the *Tænia echinococcus*, which is a Cestode or Tapeworm. Like the other Cestoda, it consists of a head and of segments, called *proglottides*. The head has a proboscis or rostellum at its end, four suckers, and a double row of hooklets. The proglottides have a water-vascular system, and the mature ones have a complete set of both male and female generative organs. The *Tænia echinococcus* is very small, and never has more than three proglottides at a time, and only the last of these is mature. It inhabits the intestine of the dog, and its life-history is as follows. The mature proglottis becomes detached, and is passed per anum, and the ova may be swallowed by man with uncooked vegetables, like watercress and lettuce, or in water; or by sheep with grass. In the stomach an embryonic head develops from the ovum and works its way to the liver, muscles, etc. Here a cyst, called a hydatid, grows from the embryonic head. If, in turn, a dog should eat a sheep's liver which contained a hydatid, the embryonic head would become attached to the dog's intestine, the cyst would fall off, and proglottides would grow from the head, and so a perfect tapeworm would be formed.

A hydatid cyst consists of an *outer coat*, which is hyaline, laminated, and elastic; and an *inner*

coat composed of embryonic cells. From these cells new echinococci heads may be formed, and from each of these new heads a cyst may grow, so that secondary cysts may be contained in the original one. The fluid of a hydatid cyst is clear, colourless, and of low specific gravity, and contains much common salt and no albumen. Hooklets detached from the secondary heads are usually found in it. Outside the true cyst is a fibrous capsule, derived from the connective tissue of the liver.

Symptoms and Physical Signs of Hydatid of the Liver.—There is no pain unless the cyst be so large as to drag on the ligaments of the liver, or to compress the neighbouring organs. If the cyst be superficial, it forms a localized projection on the surface of the liver. This projection fluctuates, and transmits a peculiar jelly-like quivering, called the hydatid thrill, or fremitus, if it be lightly touched with the fingers of one hand while it is tapped by the fingers of the other. It is not tender. If the cyst be deep-seated, only a painless enlargement of the liver may be detected, and a diagnosis can only be made if the characteristic fluid be found with the exploring-needle. Jaundice and portal obstruction are not often caused by hydatids.

Terminations.—The hydatid may die spontaneously, the contents drying up to a putty-like mass, and the capsule becoming fibrous or calcareous. Or the cyst may *suppurate* (in which case the parasite will die, if it be not dead already), and all the symptoms and consequences of liver abscess may result. Or the cyst may go on growing and burst into the peritoneum, etc.

Treatment.—A single hydatid can easily be cured

by tapping (a slow process, as the cyst nearly always refills, for a time, before disappearing), or by free incision. Multiple hydatids must be treated in the same way, but they may grow in large numbers, and extend to the omentum, etc., until death occurs from exhaustion.

CANCER OF THE LIVER.

There are three ways in which the liver may become the seat of cancer.

1. **Primary Cancer.**—This is very rare. It occurs as a single large mass of cancer, usually in the left lobe. The growth is white and hard, and does not cause much enlargement of the liver. The variety of cancer is called *schirrho-encephaloid*, and the growth infiltrates the liver lobules.

2. **Secondary Cancer.**—This is very common. It occurs as multiple nodules all over the liver. The nodules are white and soft (often diffuent at the centre), and those on the surface of the organ become umbilicated. These growths are secondary to cancer of the intestine, stomach, kidney, etc. The variety of cancer is the same as that of the 'primary' growth, and is, therefore, often columnar epithelioma. The liver may become enormously enlarged.

3. **Cancer by extension of a Cancer of the Gall-bladder.**—This occurs as a large mass continuous with the cancerous wall of the gall-bladder. Gall-stones are usually found in these cases.

Symptoms and Physical Signs.—These are progressive weakness, emaciation, an anxious facies, and a sallow complexion, constituting the cancerous cachexia; pain in the right hypochondrium; en-

largement of the liver from the presence of a projecting nodule or nodules, which may be felt to be umbilicated; the nodules are tender, and ache for some time after being touched; jaundice is common, and becomes very deep; portal obstruction may occur, leading to ascites, but not often to hæmatemesis or enlargement of the spleen; the abdominal veins are often enlarged; epistaxis and other hæmorrhages may occur, and be very severe.

The jaundice and portal obstruction result, as a rule, from the pressure of cancerous glands in the portal fissure. Jaundice and ascites together are very characteristic of cancer. The rapid enlargement of the liver, and the presence of cancer elsewhere, may help the diagnosis materially.

Treatment.—Relieve the symptoms as far as possible, particularly the pain by morphia hypodermically.

SARCOMA OF THE LIVER.

Sarcoma is rare, but may occur in young persons. The growths are often hæmorrhagic. Clinically the cases resemble cancer, but are less painful.

SYPHILITIC DISEASE OF THE LIVER.

Syphilis may affect the liver in three ways.

1. It may cause a *uni-lobular cirrhosis*, the liver being large, hard, and finely granular.

2. It may cause *gummata*. A recent *gumma* in the liver appears as a whitish tumour, and under the microscope is seen to consist of a mass of granulation tissue, growing among the liver cells. But recent *gummata* are seldom seen, because the patient does not die at that stage of the disease.

An *old gummatous liver* is usually large and covered by peri-hepatic adhesions. Its surface is deeply depressed here and there by stellate fibrous scars ; and on cutting into these scars a cheesy nodule may be found, which is the remains of the gumma. The liver between the scars may be normal, but is more often cirrhotic and sometimes lardaceous. In the last case the liver is very large.

3. Syphilis may cause a simple lardaceous liver. The **Symptoms** of a gummatous liver are very obscure. In rare cases, nodules have been felt on the surface of the liver, have disappeared after the administration of iodide of potassium, and have, therefore, been considered to be gummata. In cases of old gummata there will probably be a history of attacks of pain and tenderness over the liver, coinciding with attacks of peri-hepatitis. Jaundice is very rare, but ascites may occur from the accompanying cirrhosis, and an enlarged spleen is common from cirrhosis or from lardaceous disease. The liver may be felt to be large, hard, and very nodular ; but the nodules are not outgrowths, but merely the parts of the liver between the depressed scars. Lastly, no further increase in size of the liver occurs under observation.

Treatment.—Mercury and iodide of potassium will cure a recent case, but have not much effect when the scars have formed.

TUBERCULAR DISEASE OF THE LIVER.

This is only of pathological interest. Minute discrete tubercles are usually found in the liver in cases of acute general miliary tuberculosis, and, very rarely, large cheesy tubercular masses have been found.

DISEASES OF THE BILE-DUCTS AND GALL-BLADDER.

Suppuration within the bile-ducts in the liver has been described with abscess of the liver. A **Catarrhal inflammation** of the common bile and hepatic ducts sometimes occurs, and is probably always associated with catarrh of the duodenum and the stomach. It is caused by exposure to cold or by indiscretions of diet.

The swelling of the mucous membrane and the secretion of mucus block the orifice of the common duct, so that the symptoms are those of *obstructive jaundice*, and the liver may be found to be somewhat enlarged. In many cases the jaundice is preceded or accompanied by distaste for food, nausea, etc., indicating a gastric catarrh. The symptoms usually pass off in about a week, but the yellow colour may persist for a long time.

Treatment.—Give the sulphate and carbonate of magnesia, so as to keep the bowels pretty freely open for forty-eight hours. Then give bismuth, and afterwards bitter tonics.

The Affections of the Gall-bladder are, practically, always caused by, or, at any rate, associated with, **Gall-stones**.

A gall-stone may vary in *size* from that of a pigeon's egg downwards. In *colour* it may be dark-brown, or green or yellow or fawn coloured, or even white. Chemically, it is composed of cholesterine and bile pigments, with, perhaps, a small quantity of lime salts. If it is nearly pure cholesterine it is pale and smooth, and though it looks hard, it is soft enough to be easily broken between the fingers or cut with a knife. Also, its

specific gravity is very low, so that when dry it may float in water. If the bile pigment is in excess it is dark and hard, and less smooth, and sinks in water. The *shape* of a gall-stone is oval, but if two lie in the gall-bladder together, a facet forms at the point of contact; and if the stones are numerous they are polygonal. If cut across, some gall-stones show concentric rings, and they may have a distinct nucleus. The cholesterine part of a gall-stone is soluble in chloroform, alcohol, etc., so that most gall-stones are nearly completely dissolved when treated with chloroform, and if the solution is allowed to evaporate the characteristic crystals can



CRYSTALS OF CHOLESTERINE.

be seen. They are colourless rhomboidal plates, with a piece cut out at one corner, and they lie in super-imposed masses.

The causes of gall-stones are unknown. They occur more frequently in females and in persons of sedentary habits who live too well.

The possible consequences of gall-stones are as follows: 1. No symptoms whatever may occur. 2. The wall of the gall bladder may become fibrous, and contract firmly on the stone or stones. In these cases a hard, painless tumour can be felt in the region of the gall-bladder, but there are no other symptoms. 3. The wall of the gall-bladder may *suppurate* and *burst* into the peritoneum, causing fatal peritonitis; or, after adhesion, into the

colon, stomach, duodenum, pleura, etc.; or externally, the stones escaping. 4. The stone may enter the cystic duct, and then the common duct, causing *biliary or hepatic colic* (see below). 5. The stone may permanently block the cystic duct, causing a *cholecyst* (see below). 6. The stone may temporarily or permanently block the common bile duct, causing *jaundice*. If the jaundice be permanent the gall-bladder and liver become very large from distension with bile. 7. The stone may set up ulceration of the common duct, and suppuration, which extends into the ducts in the liver and causes *abscesses* there. 8. In *cancer* of the gall-bladder and ducts, gall-stones are usually found; but, of course, we cannot regard them as the actual cause of the cancer. 9. A large gall-stone which has ulcerated into the duodenum or colon may cause *intestinal obstruction*.

Symptoms of Hepatic Colic.—A sudden excruciating *pain* is felt in the right hypochondrium, and radiates to the umbilicus and shoulder. There may be *no tenderness*; in fact, pressure may relieve the pain. There may be *shivering* and *vomiting*. At any moment the pain may cease, if (*a*) the stone falls back into the gall-bladder, or (*b*) reaches the duodenum, or (*c*) if it perforates the wall of the duct, in which case other severe symptoms may succeed. Within twenty-four hours of the beginning of the pain jaundice usually occurs from obstruction of the common duct.

Treatment.—Hot baths, or if they be not available, hot fomentations. Morphia hypodermically. It may be necessary to administer chloroform to procure complete anæsthesia. To prevent the formation of gall-stones, it has been recommended to

give salines regularly, particularly sodium sulphite (grs. x.), and to limit the amount of fat in the diet.

A **cholecyst** results from a permanent complete obstruction of the *cystic duct*. As a consequence of the obstruction, no bile can get into the gall-bladder, and none of the mucus secreted in the gall-bladder can escape from it. Consequently the mucus accumulates and distends the gall-bladder sometimes to the size of a cocoanut. The tumour is painless and fluctuating, and there is no jaundice, as the common bile duct is free. The fluid is, usually, colourless (because all the bile pigment originally present has been absorbed from it), opalescent and slimy, and it contains albumen and cholesterine crystals. The treatment is surgical.

DIFFERENTIAL DIAGNOSIS OF CERTAIN LIVER AFFECTIONS.

If a **Large Swelling** can be felt projecting from the liver, it may be (1) a hydatid, (2) a cholecyst, (3) an abscess, (4) a mass of cancer, (5) a gumma, or, more often, a projection of the liver between depressed gummatous scars.

The **Hydatid** will be fluctuating and painless, and the exploring needle will evacuate a clear, non-albuminous fluid, containing hooklets, and, perhaps, heads of the echinococcus.

The **Cholecyst** will be fluctuating and painless; there will be a history of gall-stones, and the tumour will be strictly in the position of the gall-bladder. The exploring needle will evacuate a slimy, opalescent fluid, containing cholesterine crystals.

The **Abscess** will be fluctuating and painful, and tender. There will be remittent fever and sweatings, and a history of intestinal ulcers or of residence abroad. The exploring needle will evacuate matter which is usually chocolate coloured.

The **Cancer** may be semi-fluctuant (from softening), and is painful and tender. It is often umbilicated. There is cachexia, but no fever. The growth increases in size rapidly. The exploring needle yields *cancer juice*. Jaundice and ascites are fairly common.

The projections caused by **gummata** are always hard, and therefore only likely to be mistaken for a hard cancer. But there is much less pain, as a rule, and other syphilitic lesions may be found. It is often, however, impossible to make a diagnosis between cancer and syphilis without waiting a fortnight or so, and treating the case with iodide of potash. At the end of that time a gumma will be smaller, a bulge of the liver between gummatous scars will be unaltered, but a cancer will be larger, from uninterrupted growth.

Multiple Small Nodules on the surface of the liver may be hydatids, abscesses, cancerous nodules, or gummata, or, if very small, they may be due to cirrhosis. As regards the first four the points mentioned above will help the diagnosis. As regards cirrhosis, there will be, probably, ascites, or hæmatemesis, a history of alcoholism, and the liver may get smaller and the spleen large under observation.

DISEASES OF THE PANCREAS.

THE only disease of the pancreas which comes within the range of ordinary diagnosis is **Cancer**.

Cancer of the Pancreas is usually schirrhous, and situated at the head of the gland. It may be felt through the abdominal wall as a hard, fixed, globular mass. If the pancreatic juice does not reach the duodenum the stools may be fatty. The growth may compress the bile-duct, causing jaundice; or the pylorus, causing dilatation of the stomach, and the intermittent vomiting of large quantities of decomposing food. There may be secondary cancerous deposits in the liver.

The pancreas may be found after death to be atrophied or fatty, or cirrhotic, etc. Also cysts have been found in it.

Of late it has been found that in a considerable number of cases of diabetes the pancreas has been diseased, most often cirrhotic. It has also been shown, experimentally, that *complete* removal of the pancreas in animals is followed by glycosuria. This result is not due to the shock or injury to the tissues which accompanies the operation, because if a small piece only of the gland be left glycosuria does not ensue. Consequently it has been suggested that the pancreas, like the thyroid gland (*vide* Myxœdema) has an internal secretion, *i.e.*, that the pancreatic cells supply some material to

the portal vein, which material is essential to the normal metabolism of sugar, so that when this material is absent, sugar is present in excess in the blood, and therefore is eliminated by the kidneys. On the other hand, cases of diabetes have been recorded in which the pancreas has been found normal; and in other cases extensive pancreatic disease has been found without glycosuria. It is even said that the results of experimental extirpation are not constant. The question, is, therefore, still unsettled.

DISEASES OF THE ALIMENTARY CANAL.

General Considerations.—Diseases of the alimentary canal may be peculiarly difficult to diagnose ; and in certain cases it may be necessary to use special means of observation. These will be referred to later, but at the outset it is essential to say a few words about the examination of the *tongue*, the *abdomen*, the *vomit*, and the *fæces*.

THE TONGUE.

The chief points to be noticed are its degree of moisture, its colour and size, and its surface.

The tongue is dry in all fevers (including inflammations), and it is dry, glazed, and small in diabetes.

A thickly-furred, or a large flabby tongue, is common in gastric catarrh ; while a tongue with red edges and swollen red papillæ indicates a more serious gastritis, unless it be merely a part of general stomatitis.

The dry, glazed, brown, tremulous tongue of the *typhoid state* is very characteristic. Much the same kind of tongue may be seen in uræmia.

Scars on the tongue are very important as indicating past syphilis : and defective movements of the tongue may be the earliest sign of paralysis.

On the whole, however, the importance of the tongue as an index of particular affections has been exaggerated.

The offensive smell of the mouth in gangrene, in intestinal obstruction, and in uræmia is important.

THE ABDOMEN.

Inspection of the abdomen may reveal the *absence of respiratory movements* as in acute peritonitis; or a *general prominent swelling*, as in ovarian cysts or pregnancy; or a *general swelling*, which is flat in front, and bulging at the sides, as in ascites; or an *irregular swelling*, as in various abdominal tumours. In the case of aneurism, *pulsation* may be seen. In cases of chronic intestinal obstruction, with hypertrophied intestinal walls and distended intestines, the movements of the bowel in peristalsis may be conspicuous. These visible movements are called *tormina*.

The enlargement of the superficial abdominal veins may be important as indicating pressure on the inferior vena cava.

Percussion of a normal abdomen should yield a deep resonant note from the ribs to the pelvis, and across from one erector spinæ to the other. Over the stomach the note is louder, because there is a larger cavity containing air. If the stomach or intestine be made tense by inclosed flatus, the note is higher pitched, or *tympanitic*. By marking the area of this high-pitched note, a distended stomach, or sigmoid flexure, may be mapped out.

General distension of the intestines is called *tympanites*, and is most common in peritonitis, acute or chronic, and in intestinal obstruction.

Tumours and inflammatory thickenings of the wall of the stomach or intestine do not, as a rule, give a dull note, but high-pitched resonance. On the other hand, swellings of all kinds of the abdominal wall, or peritoneum, fluid accumulations within the peritoneum, and all enlargements of the abdominal and pelvic viscera, may cause large areas of dulness.

Palpation of a normal abdomen should reveal practically nothing. Palpable enlargements of the liver, spleen, kidneys, pancreas, pelvic viscera, and abdominal wall need not be considered here, except to say that the pulsation and thrill of an aneurism may be felt. The stomach may be indistinctly felt if its walls be hypertrophied; but any palpable swelling distinctly forming part of the stomach, either at the pylorus or elsewhere, is pretty sure to be a cancer. Such tumours have often a pulsation communicated to them by the subjacent aorta.

As regards the Intestine : In chronic obstruction the hypertrophied bowel may be felt as well as seen; in perityphlitis and dysentery the inflammatory thickening of the gut may make it palpable; and in intussusception the triple layer of intestinal wall forms a definite tumour. All these four conditions cause *sausage-like* tumours. Cancers of the intestine may also be felt through the abdominal wall, and if large they may be sausage-shaped.

As regards the peritoneum, cancerous nodules and tubercular glands are often palpable. The omentum in particular, both in cancer and in chronic peritonitis, can often be felt as a transverse band in the lower epigastrium.

Auscultation is not of great assistance in abdominal affections. In acute peritonitis friction sounds can be heard when the surfaces of the

viscera, roughened by fibrin, rub against each other. Splashing sounds can also be heard when the stomach is *dilated*, and filled with fluid and gas; and it is said that the trickling of fluid through an œsophageal stricture may be audible and of diagnostic importance. The bruit of an aneurism can of course be heard.

VOMITING.

Vomiting may be of three kinds :

1. **Cerebral vomiting**, *i.e.*, vomiting caused by diseases of the brain, especially tumours, or by poisons circulating through the bloodvessels of the brain, such as the toxins of the fevers, uræmia, such drugs as apomorphia, etc.

2. **Gastric vomiting**, caused by various conditions of the stomach, such as foreign bodies within it, or improper food, or irritant poisons introduced into the stomach; or by diseases of the stomach-wall, or by obstruction of the pylorus, etc.

3. **Reflex vomiting**, in which the act of vomiting is excited by a reflex process, which originates in irritation of the throat or the uterus (*e.g.*, pregnancy), or ovaries or kidneys (*e.g.*, calculi), etc. Vomiting may even be excited by unpleasant smells or sights.

The differential diagnosis of these three kinds of vomiting is usually easy, because other symptoms, pointing to the brain, stomach, etc., are usually present.

The characteristics of *gastric* vomiting, however, are that there is usually great *nausea*, which is, at least for a time, *relieved* by the vomiting. The vomiting, again, often follows the taking of food,

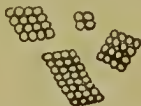
and the vomited matter may present peculiarities. It is always important to note the time after food at which vomiting occurs; because in pyloric disease it may occur some hours afterwards, while in disease of the cardiac end it follows very quickly.

The Examination of Vomited Matter.—The *quantity* is important, as if great it indicates a *dilated* stomach. If *unaltered proteid food*, such as meat, be present, it indicates either that this food has recently been swallowed, or that no attempt at digestion has been made. The absence of *free hydrochloric acid* is said to indicate *cancer* of the stomach. The presence of *bile* indicates violent retching, or an obstruction of the intestine below the orifice of the common bile-duct. Bile may be recognised by the yellow or green colour, by the bitter taste (to the patient), and by chemical tests (see Diseases of the Kidney). A large quantity of *mucus* indicates gastric catarrh, and is often seen in the morning sickness of the alcoholic.

The vomit may also contain *blood* (see below); or *pus*, from a liver or other abscess; or *feculent material*, as in intestinal obstruction, when it is called *stercoraceous*; or *gall-stones*; or *worms*, chiefly *Ascarides*; or foreign bodies and poisons which have been swallowed. With the *microscope*, striped muscular fibres, elastic fibres, fat globules, and starch granules, all derived from the food, may be seen; and, in certain cases, epithelial and pus cells, and red blood corpuscles can be detected. *Torulæ* and *sarcinæ* are sometimes present, and they indicate that the food has been a long time in the stomach, and is fermenting. Consequently, they are most often found in dilatation of the stomach due to

pyloric obstruction, and the vomit is abundant, watery and frothy.

Torulæ are yeast-fungi, about the size of a red blood corpuscle, but oval in shape. Their protoplasm may be vacuolated. They may occur singly or arranged in a string like a necklace of beads.



SARCINÆ VENTRICULI.

Sarcinæ are also fungi, and belong to the class called *Schizomycetes*. They are, in fact, micrococci, arranged, usually, in groups of eight. Each group, therefore, looks like a small cube with a cross on each face, and a collection of such groups has the appearance of a miniature woolpack.

Vomiting of Blood is called *Hæmatemesis*.

Causes of Hæmatemesis.—Very acute *gastritis*, such as follows corrosive or severe irritant poisons; *ulcers* of the stomach, particularly the *perforating ulcer* and *cancerous ulcers*; *cirrhosis of the liver*; and, more rarely, other forms of portal obstruction; *blood conditions*, such as occur in *malignant anæmias*, *acute yellow atrophy of the liver*, *yellow fever*, *typhus*, and other malignant fevers, *uræmia*, *scurvy*, etc.; the bursting of an *aneurism* into the stomach, or œsophagus; vicarious menstruation; the swallowing of blood effused from the nose or lungs.

Diagnosis of Hæmatemesis.—If it has been long in the stomach, the blood looks like *coffee-grounds*, and under the microscope the corpuscles are shrivelled. More commonly the blood is merely dark

from the action of the acid in the gastric juice. The blood may be mixed with food. In many cases the faintness from loss of blood precedes the vomiting. Other symptoms referring to the stomach or liver may be found. In *hæmoptysis*, it will be remembered, the blood is bright red (from oxygenation), frothy, and mixed with sputum, and symptoms and physical signs referred to the lungs will be found. Further, as the blood cannot stay in the bronchial tubes, the faintness is synchronous with the hæmoptysis; and, lastly, there will be blood-streaked sputum coughed up for some time after the first gush of blood. If the patient be watched it may be easy to see whether he is coughing up the blood or vomiting it.

Treatment of Hæmatemesis.—Insist on absolute rest; give ice to suck, and put an icebag to the epigastrium. Sometimes a mustard-plaster to the epigastrium is useful. Prescribe acid. gallic. grs. x., and tiuct. opii ℞. in ℥i. of aq. chloroform. Other useful drugs are ergot and dilute sulphuric acid and turpentine (if no ulcer is suspected). It may be necessary to feed by enemata only, for some days. Do not give brandy unless the faintness is very marked. Later on give tonics of all kinds.

EXAMINATION OF THE FÆCES.

Under the microscope fæces may be seen to contain the débris of food, still recognisable as such (see Vomiting), and crystals of the ammonio-magnesian phosphate are common. Macroscopically the fæces may be shaped like a *pipe-stem*, or a *riband*, as in *stricture of the rectum*; or they may contain *tapeworm proglottides*, *threadworms* or

roundworms, or *gall-stones*. If they contain undigested food, the condition is called *lientery*, and it is due to disease high up in the small intestine. In *obstructive jaundice* the stools are pale, clayey, and offensive. Bits of *polypi* or other *tumours* may be passed per rectum; and in cases of *mucous polypus* and of *columnar epithelioma*, a whitish soft jelly-like material may be mixed with the *fæces*. In intestinal obstruction, due to *intussusception*, no *fæcal* matter is passed, but only *blood* and *mucus*. Mucus is also abundant in the stools of catarrhal enteritis; and pus may be found in more severe enteritis, or if an abscess burst into the bowel. The causes of *Melæna* (blood in the stools) are very acute enteritis: ulceration of the bowel, especially in typhoid fever, ulcerative colitis, and dysentery; but also in cancerous and tubercular and other ulcers: portal congestion, especially in cirrhosis and mitral disease, piles being often present: tumours of the bowel like polypi or cancers: blood conditions (for which see hæmatemesis): intussusception: rupture of aneurism: traumatism: hæmorrhage into the stomach passing on into the bowel. The blood may be dark, like tar, if it has been long in the bowel.

The treatment of *Melæna* is much the same as that of hæmatemesis. But in addition the sulphate of copper (gr. $\frac{1}{2}$) and acetate of lead (gr. ii.) have been recommended, either of them in a pill with opium. If the seat of the hæmorrhage be near the anus, astringent enemata can be tried.

Diarrhœa.—The causes of diarrhœa are: severe and catarrhal enterites of all kinds, such as are excited by irritant poisons, purgatives, improper food, cold, septic diseases, etc.; cholera; typhoid

fever, ulcerative colitis, dysentery, and other cases in which ulceration occurs ; lardaceous disease ; mental emotion.

Certain types of diarrhœa may be mentioned.

(1.) In **Enteritis** the stools are large in quantity, offensive, watery, brownish, with, perhaps, much mucus intimately mixed with them. They are discharged violently from the bowel after a griping pain in the abdomen.

(2.) In **Cholera** they are large in quantity, pale and opalescent, like water in which rice has been boiled. They contain the comma-shaped bacillus. At first they are discharged violently, but afterwards run away through the relaxed anus.

(3.) In **Typhoid** they are large in quantity and yellow and pultaceous, like split-pea soup. They are offensive, and are passed easily. They contain typhoid bacilli.

(4.) In **Dysentery** the stools may be large in quantity, but when the motions are very frequent they are small, and consist of blood and mucus, with often pieces of mucous membrane or of fibrinous false membrane. If the ulceration is all over the colon, the fæcal matter is intimately mixed with the discharge ; but if the ulcers are only in the sigmoid flexure and the rectum, hard lumps of fæces are present in the mucus. If there is much sloughing the stools are very offensive. The pain is felt along the course of the colon, and there is constant tenesmus or desire to empty the rectum. In bad cases, after great pain and straining, only a small motion is passed without any relief to the tenesmus.

DISEASES OF THE MOUTH.

INFLAMMATION OF THE MOUTH— STOMATITIS.

Varieties.—1. Simple; 2. Follicular; 3. Ulcerative; 4. Aphthous; 5. Parasitic; 6. Gangrenous or Cancrum Oris.

In **Simple Stomatitis**, red, swollen patches are seen all over the mouth, and the gums are also red and swollen, and bleed readily. The mucous membrane is hot, but there is usually a good deal of secretion. The affection is most common in children, and is due to want of care in cleansing the mouth. In adults it may be due to carious teeth, mercurial poisoning, scurvy, etc. In more severe cases the follicles in the mouth become distended, forming little yellow projections (like split peas), which eventually burst, leaving little round ulcers. The affection is then called **Follicular Stomatitis**. In cases which are still worse the round ulcers spread in extent and coalesce, forming large irregular ulcers. This is **Ulcerative Stomatitis**.

In **Aphthous Stomatitis** whitish-yellow spots are found on the inflamed mucous membrane. Some think that they are vesicular, others that they are fibrinous.

In **Parasitic Stomatitis** or **Thrush** gray or white patches are found which consist of dead epithelium and the spores and mycelium of a fungus called *Oidium Albicans*. The patches are easily detached.

In all cases of Stomatitis there is a little fever, and the mouth is very painful. There may also be gastro-intestinal disturbance.

Treatment.—Give a mild aperient and then

tonics, such as quinine and iron, with chlorate of potash. Local treatment is of most importance, especially in infants. In simple cases wash out the mouth most thoroughly with glycerine of borax, or solutions of chlorate or permanganate of potash. In ulcerative cases, glycerine and sulphurous acid in equal parts make a good wash, and sometimes the stick of nitrate of silver must be applied to the ulcers.

Gangrenous Stomatitis—Cancrum Oris—Noma.—This disease is most common in children who are debilitated by some disease (very often measles) or by bad feeding. A hard nodule appears in the mucous membrane of the cheek, and rapidly ulcerates into the mouth. The ulcer is gray on the surface, and has a hard base and hard, clean-cut edges. It extends very quickly. Outside, the cheek is at first deep red, and brawny to the feel. Then a black speck appears in the middle of the red area. This is a slough, which becomes detached, and leaves a hole right through the cheek into the mouth. From the time of ulceration the breath becomes very offensive, and death usually occurs in a few days. There is very little pain.

Treatment.—Put the child under chloroform, and either apply strong nitric acid freely, all over and round the ulcer, or scrape freely with a curette. Internally give tonics.

ULCERATION OF THE MOUTH.

In addition to the ulcers mentioned above, the following may occur in the mouth :

1. **Ulcers from Jagged Teeth.**
2. **Tubercular Ulcers,** usually on the sides or beneath the anterior part of the tongue, and irre-

gular in outline. Bacilli may be found in the discharge.

3. **Syphilitic Ulcers.**—Secondary ulcers may occur on the inside of the lips or cheek, or on the sides of the tongue. They tend to be sinuous in outline and not very deep. Tertiary ulcers and gummata occur on the dorsum linguæ. The effects of treatment are of diagnostic importance.

4. **Cancerous Ulcers.**—These occur on the lip or tongue, and are ulcerations of a projecting nodule. The kind of cancer is squamous epithelioma, and by microscoping a scraping of the projection, the diagnosis can be made certain.

5. **Variolous.**—These are seen after the suppuration and bursting of a pock in the mouth.

INFLAMMATION OF THE TONGUE— GLOSSITIS.

This is usually caused by an accidental scratch or puncture, which becomes septic. The tongue in a few hours swells to an enormous size, so as even to threaten suffocation. It may also protrude from the mouth. At the same time fever and other signs of septicæmia are present. The treatment consists in incising the tongue freely, and giving a calomel purge and quinine.

DISEASES OF THE FAUCES.

ACUTE TONSILLITIS.

Causes.—Inflammation of the tonsils occurs in many specific fevers, such as scarlet fever, diphtheria, etc., and the tonsils may be affected by syphilis, tubercle, and cancer ; but tonsillitis may occur independently, and in such circumstances it has been attributed to cold, to bad air, and other indefinite causes. Simple acute tonsillitis used to be called *quinsy*. One attack predisposes to another, and the condition often becomes chronic.

Symptoms.—There is distinct fever, often ushered in by shivering, and accompanied by all the pyrexial symptoms, dry skin, scanty urine, etc. There is pain in the throat, and *swallowing* is extremely painful. When looked at, the tonsils are very red and swollen, and may even block up the whole of the fauces. In bad cases lacunar or follicular abscesses may form, appearing as small yellow projections which burst, and leave round ulcers. Sometimes the whole tonsil suppurates, and may burst, leaving a deep ragged ulcer. The enlarged tonsil may be felt, externally as a tender swelling behind the jaw, and the neighbouring lymphatic glands are large, painful, and tender, being affected by acute lymphadenitis. The speech is thick, and the patient may have to breathe through the mouth, and consequently snores during sleep. In most cases the swelling of the tonsils subsides gradually, but if an abscess forms and bursts, relief is more sudden.

CHRONIC TONSILLITIS.

This usually follows repeated attacks of acute tonsillitis, or stomatitis or other forms of irritation of the mouth or fauces.

Symptoms.—There may be some discomfort on swallowing, but more often there is merely general ill-health, with perhaps an irritative cough. The patient breathes through the mouth, and consequently the mouth is kept open, and there is snoring at night. On examination one or both tonsils are large, irregular on the surface, and not particularly red. The patient may be deaf.

Treatment.—In acute cases give calomel grs. iii., with pulv. jalap. grs. x. at once to an adult, or a smaller dose of the same powder to a child. Then give quin. sulph. grs. v. with tinct. ferr. perchlor. ℥ xv., potass. chlorat. grs. x., and acid. hydrochlor. dil. ℥ v. in an ounce of water every sixth hour. Locally, let the patient inhale steam, and gargle with chlorate of potash or Condy's fluid. Painting the fauces with glycerine of tannin or perchloride of iron, etc., is useful, especially in chronic cases. The tinct. guiaci ammon. in ℥ xx doses is especially recommended by some authorities; or tinct. aconiti ℥ i. in water every hour. In bad cases with much swelling and tension the tonsil may be incised. In chronic cases the tonsils should be removed, and cod-liver oil, maltine, etc., administered.

FAUCITIS—INFLAMMATION OF THE FAUCES.

It is convenient to call by this name an inflammation of the soft palate, uvula, and pillars of the fauces. It is nearly always present in tonsillitis,

but may occur without enlargement of those glands. In many cases pharyngitis is also present.

In acute cases the fauces are red, swollen, granular and secrete freely. There is pyrexia, with often an irritative cough. In chronic cases the parts are paler and œdematous, particularly the uvula, which may be so elongated as to irritate the larynx. Chronic faucitis is known popularly as a *relaxed throat*. It may be due to excess of alcohol or tobacco. The treatment is the same as that of tonsillitis. The irritability of the parts may be allayed by opium (in a lozenge or linctus), or by spraying with cocaine solution, or by sucking ice. An œdematous uvula may be clipped with advantage.

PHARYNGITIS.

Pharyngitis occurs under the same conditions as faucitis. The acute inflammation which follows the swallowing of corrosives or foreign bodies will be considered with the diseases of the Œsophagus, but two special affections must be mentioned here. One is **Pharyngitis Sicca**, which is a form of chronic pharyngitis in which the pharynx is dry, not much swollen, red, and beefy-looking. On its surface there is often a gray mass of dried phlegm. Its causes are doubtful, and it is best treated with iodide of potash internally and glycerine locally. The other affection is **Post-nasal Adenoids**. In this, as a result of chronic faucitis or pharyngitis, there is a growth of granulation tissue with proliferation of the mucous gland-acini around the posterior nares. These growths are red, soft and spongy, and project considerably. They block the posterior nares, causing the patient to breathe through the

mouth and to snore. They also cause deafness by blocking the Eustachian tube. They must be scraped away with a curette, or by the finger-nail.

DISEASES OF THE ŒSOPHAGUS.

ŒSOPHAGITIS.

Causes.—Practically acute œsophagitis results either from the swallowing of boiling water or corrosives, like the mineral acids, carbolic acid, etc.; or from the impaction of a pin or fishbone which has been swallowed with the food. In the latter case the puncture becomes septic, and an intense inflammation results.

Symptoms.—There is a burning pain along the œsophagus which is very intense during the passage of food down the tube. There is tenderness on pressing the larynx backwards. In cases of poisoning the symptoms of acute gastritis (see p. 48) are present, and sloughs of mucous membrane may be regurgitated. In the other cases there may be general pyæmia.

Terminations.—In the cases of corrosive poisoning, if the patient survive, a fibrous stricture of the œsophagus may result when cicatrization occurs.

Treatment.—Ice may be sucked and the diet must be liquid. It may be necessary to feed exclusively by the rectum. A mixture containing the subnitrate of bismuth in mucilage is useful. As soon as cicatrization has occurred, the bougie can be passed daily to prevent contraction.

ŒSOPHAGEAL DYSPHAGIA.

By this term is meant difficulty of swallowing due to the œsophagus. The causes are acute œsophagitis, paralysis of the œsophagus, and œsophageal obstruction.

Paralysis of the Œsophagus may occur in diphtheria, in bulbar paralysis, hysteria, etc. Any solid food which has passed the pharynx may remain for a long period in the œsophagus, causing great discomfort. A bougie can be passed with ease.

Œsophageal Obstruction may be caused by :

- (a) Things inside the Tube, such as false teeth or coins, etc.
- (b) Changes in the Wall, such as cicatrization, after corrosive poisoning; cancer; syphilitic disease; etc. This group constitutes Œsophageal Stricture, and must include Spasmodic Stricture.
- (c) Pressure on the Œsophagus from without, as by an aneurism or mediastinal tumour.

Symptoms of Œsophageal Obstruction.—In cases of organic stricture and of pressure on the œsophagus from without, the symptoms come on gradually. First solids cannot be swallowed, then liquids. The patient may point out a particular spot on the chest, at which he feels that the food is arrested; and there is pain and discomfort there until it is regurgitated, or passes the obstruction. If the food is regurgitated, it is in the same condition as when swallowed, that is, it is not acid from the action of the gastric juice. Sometimes in chronic cases the food may be regurgitated violently on account of hypertrophy of the muscular coat of the

œsophagus. There is always emaciation from starvation. If a bougie be used, the position of the stricture and the size of its aperture may be ascertained, but of course no attempt to pass the instrument must be made until careful examination has excluded the presence of an aneurism. In old cases the œsophagus is much dilated above the stricture. In Cancer the symptoms are as above, but there is more pain, and the regurgitated material may be bloodstained, or blood may be found on the bougie. Sometimes the stricture is suddenly relieved by the detachment of necrosed pieces of the growth. Cancer of the œsophagus is nearly always squamous epithelioma, and usually is situated opposite the bifurcation of the trachea, or behind the cricoid cartilage. Death may often occur from the cancer extending into the trachea or pleura, and causing pneumonia and gangrene of the lung. Sometimes the growth extends to the pericardium, causing pericarditis.

Spasmodic Stricture of the Œsophagus is called **Œsophagismus**. It is most common in women or neurotic men. The symptoms of œsophageal obstruction are intermittent, *i.e.*, at one time not even fluids can be swallowed, while at other times solids pass easily. There is no pain and there may be no wasting. On passing a bougie steady pressure usually overcomes the obstruction, and a peculiar gripping of the bougie by the spasmodically contracted gullet may be felt. A good-sized tube may be passed as easily as, or more easily than, a small one.

Treatment.—If there be *cicatrization*, the habitual passage of the bougie will, probably, effect a cure. In *spasm* the same treatment is good, but

the chief thing is to give the patient confidence, and, if possible, teach him to pass a bougie himself. Antispasmodics, such as bromides, may be tried. In *cancer* never use a bougie as such, once the diagnosis is made, but try cautiously to insert a Symonds' tube. If this cannot be done, or the patient cannot tolerate it, perform gastrostomy. In aneurism, gastrostomy may also be necessary. In all cases, except spasm, nutrient enemata may be given with advantage while awaiting the results of other treatment.

DISEASES OF THE STOMACH.

CONGESTION.

Causes.—Active congestion of the stomach may be considered as the early stage of gastritis. Mechanical congestion results from portal obstruction, and therefore occurs in cirrhosis of the liver, and when malignant or other tumours compress the portal vein. It also occurs as part of the general congestion in mitral disease.

Pathological Anatomy.—The mucous membrane is swollen and dark red, and, as catarrh is usually also present, the surface is bathed in mucus. Sub-mucous petechial hæmorrhages may be seen.

Symptoms.—There is loss of appetite, dyspepsia, and often nausea, with epigastric discomfort. In extreme cases *hæmatemesis* may occur.

Treatment.—Treat the cause when possible. In most cases saline and watery purgatives are useful, and diuretics may be given.

INFLAMMATION OF THE STOMACH— GASTRITIS.

Gastritis may be acute, subacute, and chronic.

Acute Gastritis.

Causes.—Corrosive and irritant poisons, like the mineral acids, perchloride of mercury, arsenic, antimony, etc. ; other irritants, such as bad food or decomposing sputum (when swallowed), and mechanical irritation from foreign bodies, such as coins or powdered glass.

Pathological Anatomy.—The mucous membrane is bright red, swollen, and covered with muco-pus. There may be patches of false membrane on it, or it may be ulcerated.

Symptoms.—If a poison has been taken there is œsophageal dysphagia, and the mouth and pharynx may exhibit acute inflammation. As regards the stomach itself, in all cases there is great epigastric pain and tenderness. Vomiting is urgent, and the vomit may contain mucus, pus, and blood. There is pyrexia with a small pulse, and extreme thirst. The face is anxious, and exhaustion sets in early. All the symptoms of peritonitis (p. 84) may follow, and when the irritant reaches the intestines diarrhœa occurs.

Terminations.—If the patient does not die the gastritis may become chronic, with fibrous thickening of the wall of the stomach.

Treatment.—Give morphia hypodermically, apply hot fomentations to the epigastrium, and give gruel, barley-water, or milk with lime-water. Ice may be sucked, and raw eggs are soothing. Later on bismuth can be given, as in subacute cases.

If the case be one of poisoning, the stomach-pump can only be used, if the poison be a comparatively mild irritant. As regards antidotes: For *arsenical preparations* use hydrated peroxide of iron; for *tartar emetic* use tannin, or substances containing it, such as tea; for *mercurial* or *copper* salts use albumen, such as white of egg; for *nitrate of silver* use common salt; for *mineral acids* use magnesia or an alkaline carbonate; for *oxalic acid* use magnesia or chalk, or the plaster from the ceiling, but not alkalies, as the alkaline oxalates are poisonous; for *caustic alkalies* use vinegar.

Subacute Gastritis.

Causes.—Cold, over-eating, and alcoholic excess. Causes which, in an adult, may only produce indigestion, may set up gastritis in a child.

Pathology.—The inflammation is a catarrhal one (like all subacute inflammations of mucous membranes); that is, there is dilatation of the bloodvessels, abundant secretion of mucus, and a moderate exudation of leucocytes (see the Pathology of Bronchitis).

Symptoms.—These are much the same as those of the so-called *bilious attack*. There is moderate pain and tenderness at the epigastrium. Nausea is most marked, and vomiting is frequent. The vomit contains much mucus, but after a time becomes bilious. There is often headache and thirst; the tongue is foul, and, of course, the appetite is completely in abeyance. The urine is febrile, and particularly rich in urates. If the catarrh extends to the bowel, diarrhoea follows the stomach symptoms, but if not, constipation is present. Most cases last

only for a day or two, but in children and debilitated subjects the affection may persist for two or three weeks, and perhaps become chronic.

Treatment.—If the vomiting be urgent, try to make the patient swallow large quantities of warm water, for if the water is vomited the stomach is washed out, and relief is obtained. If the case is seen early, give a blue pill, and follow it with a saline aperient. But if the vomiting has lasted some time (so that the stomach is clear), give tinct. opii, ℥x.; bismuth. subnit., grs. x.; mucilag. tragacanth., ʒss.; and aq. anethi, ʒss., every four hours. Or try small doses of acid. hydrocyan. dil. in an effervescing mixture. The diet must be fluid, milk and lime-water or milk and soda-water being the best things. Slight counter-irritation to the epigastrium may be of great service.

Chronic Gastritis—Chronic Gastric Catarrh.

Causes.—Prolonged mechanical congestion, as in cirrhosis of the liver, and old cases of mitral disease, chronic alcoholism, chronic indiscretions of diet, and uræmia. It may be a sequel to an acute attack.

Pathological Anatomy.—After death the mucous membrane is found to be thick and cedematous-looking, purple in colour, and covered with mucus. What has been happening is a constant dilatation of the vessels, and a constant proliferation of the epithelium of the glands, and, therefore, a constant hypersecretion of mucus. There is also a slow exudation of leucocytes. In some cases granulation tissue develops in the submucous connective tissue, and when the granulation tissue becomes fibrous (as it must do in time), the wall of the stomach

becomes thick, firm, and crisp. Consequently, the stomach does not collapse when opened, and, from the contraction of the new fibrous tissue, the whole organ becomes small and distorted. A similar distortion of the stomach may be seen in a rare form of chronic peritonitis, when the peritoneum over the stomach is involved, only then the new fibrous tissue is subperitoneal, and not submucous.

Symptoms.—In discussing the symptoms of acute gastritis, dyspepsia was not mentioned, as the patient was scarcely capable of taking food; but it will be understood that in that affection digestion does not go on. But in chronic gastritis dyspepsia is, strictly speaking, present, and the taking of food excites nausea, epigastric discomfort, and sometimes vomiting. There may be complete anorexia and loathing of food, meat in particular being disliked. The symptoms are particularly marked in the morning, because the patient wakes with his stomach covered by mucus, and is uncomfortable until it is vomited or has passed into the duodenum. There is always mental depression, and emaciation may occur.

Treatment.—Alcohol must be avoided, a simple diet must be ordered, and exercise insisted on. A glass of milk or cup of tea should be taken before getting out of bed. In bad cases it is useful to give the bismuth mixture (see p. 50) before rising; and if the nausea be great, and there is no constipation, this may be continued three times a day. If there be constipation the magnesia mixture is better. If the nausea be not marked during the day, the anorexia may be treated with *nux vomica* or other bitters; and even a few drops of *spir. ammon. aromat.* may be given as a stimulant.

ULCERATION OF THE STOMACH.

The stomach may be the seat of *follicular* ulcers like those of the mouth, and ulceration is common in cancer. Syphilis and tuberculosis may also possibly give rise to ulceration. But there is one special form of gastric ulceration called the **perforating ulcer**.

Perforating Ulcer of the Stomach—Causes.—It is most common in women, and particularly in anæmic women. The lesion is believed to take place as follows: One of the arteries supplying the stomach becomes occluded by an embolus or thrombus. Immediately the piece of stomach-wall supplied by that artery becomes anæmic, and its vitality is consequently so reduced that it cannot resist the action of the gastric juice, but becomes digested out, leaving an ulcer. If the whole thickness of the wall be digested perforation results.

The reasons for believing that the above is the process which takes place, are: 1. That the ulceration may occur with extreme rapidity, and we know that the results of embolism are always rapid, and so is the digestive process. 2. The funnel-shape of the ulcer corresponds with the shape of the piece of tissue supplied by a gastric artery. 3. The smoothness of the wall of the ulcer is more like the result of a solvent process than of ordinary ulceration. 4. In certain cases an embolus has been found. 5. Experimentally, ligature of one of the gastric arteries produces the same kind of ulcer.

Failing this embolic theory, we must suppose that in anæmic persons a small portion of the stomach may become absolutely anæmic accidentally (as by the pressure of an undigested bolus

of food lying on it), and in that condition may be dissolved by the gastric juice.

Pathological Anatomy.—The ulcers are most commonly found at the pyloric end, along the lesser curvature, and on the posterior wall. They are funnel-shaped, with the apex at the peritoneum, and the base at the mucous coat. Their walls are smooth, and they present, as a whole, a clean-cut appearance. They may be multiple. If perforation has occurred, peritonitis will be present; but sometimes adhesions to the liver, pancreas, etc., may have formed before perforation occurred, and then fistulæ or abscesses may be found connected with the hole in the stomach. In old cases there is great fibrous thickening of the edges of the ulcer, and if cicatrization occurs there may be enough contraction to constrict the pylorus or distort the stomach, which may even become hour-glass shaped. The ulcers which result from *post-mortem* digestion of the stomach are quite different in appearance. They are superficial, and have soft, ragged edges. They are, consequently, ill defined, and the mucous membrane around is soft and jelly-like.

Symptoms.—These are very obscure, and, unfortunately, a fatal hæmatemesis or perforation may occur without any symptoms but those of slight dyspepsia having preceded. A localized *pain* and *tenderness* are usually present in the epigastrium, and perhaps also in the back. Severe pain immediately after swallowing food, particularly hot food, is common. This pain may excite vomiting, after which it is relieved. There may be milder symptoms of dyspepsia, such as mere discomfort after food, heartburn, or flatulence. Pyrosis (the regurgi-

tation of clear acid liquid) is usually complained of. The hæmatemesis may be moderate or very copious. If perforation occurs, the symptoms of peritonitis (see p. 84) will follow; but death may be instantaneous from shock, due to the escape of a large quantity of food.

If the ulcer becomes chronic, the symptoms persist, and those of pyloric obstruction (see p. 55) may be added. The patient usually becomes thin, sallow, and cachectic.

Treatment.—If you are certain that there is a recent gastric ulcer, especially if hæmatemesis has occurred, stop all food by the mouth and give nutrient enemata. For the treatment of hæmatemesis, see p. 35. In less urgent cases the diet must be liquid, and the stomach soothed as much as possible by bismuth or effervescing mixtures, with opium or dilute hydrocyanic acid. In still less urgent cases iron must be given. It must be remembered that the stomach symptoms in chlorosis closely resemble those of gastric ulcer, and that the former may sometimes be rapidly cured by reduced iron in 5 grain doses. Constipation should be treated by enemata only. In giving nutrient enemata the rectum must be emptied of fæces first, and then 4 oz. of fluid, at a temperature of 100° F., must be slowly injected. The injection may be repeated every four hours. Milk and beef-tea can be used, but should be peptonized or treated with pancreatic extract first. Raw meat-juice makes a good nutrient enema when there has been much hæmorrhage. Nutrient suppositories are now made, and may be tried.

DILATATION OF THE STOMACH—PYLORIC OBSTRUCTION.

Dilatation of the stomach is usually due to obstruction of the pylorus, and is preceded by hypertrophy of the muscular coat of the stomach. But in a few cases the muscular coat becomes fatty or atrophied, and then the stomach may dilate without any pyloric obstruction.

The Physical Signs of a Dilated Stomach are: An increase in the area of stomach resonance which may displace the heart's dulness above, the splenic dulness posteriorly, and, below, may reach nearly to the pubes: on auscultation splashing may be heard on shaking the patient, or if he is made to swallow water during auscultation, it may be heard to fall into the stomach with a noise as if falling into a well: if vomiting occurs, the quantity is large, and the vomit contains *sarcinæ*.

Causes of Pyloric Obstruction:

1. **Foreign bodies impacted in the pylorus**, such as coins, corks, and various substances accidentally swallowed.
2. **Stricture of the pylorus.** Most often this is caused by cancer, but sometimes by cicatrization after some form of gastric ulcer.
3. **Pressure on the pylorus from without** by cancer of the pancreas, liver, etc., or by aneurism.

Symptoms of Pyloric Obstruction.—These nearly always come on gradually. At first there is distension, with discomfort or actual pain an hour or two after food, when the food should be passing through the pylorus. These symptoms lead to vomiting, which relieves them. Later on, as the stomach

dilates, the pain, etc., and the vomiting occur less often (perhaps every three days), because the stomach can hold more without contracting on its contents. But, on the other hand, the quantity vomited each time becomes much greater. In fact, the stomach may, without apparent effort, eject two or three bowlfuls of decomposing food which has undergone stomach digestion, and contains sarcinæ. There is, of course, never any bile in the vomit. At the same time the physical signs of a dilated stomach can be obtained, and if a tumour can be felt in the pyloric region the diagnosis may be considered complete.

Treatment.—The operative measures consist in excising the pylorus, or dilating it through an opening in the stomach, or establishing a communication between the stomach and jejunum. Failing these, very great relief can be given by washing out the stomach with water or dilute antiseptic lotions. If this be done systematically, the food does not remain and decompose in the stomach, and so the irritation resulting from its decomposition does not occur. And, in addition, each new meal is put into a clean stomach, and a certain amount of it may be absorbed. If the stomach cannot be washed out, an attempt may be made to limit the decomposition by giving antiseptics like creasote or carbolic acid. Rectal feeding usually becomes necessary.

CANCER OF THE STOMACH.

Pathology.—The growth is most commonly situated at the *pylorus*, but may be on the *lesser curvature*, or at the *cardiac orifice*, or it may be part of a *peritoneal cancer*.

Pyloric Cancer is most often scirrhus, but may

be encephaloid, or columnar epithelioma. In the last two cases it may undergo colloid degeneration. All cancers in this position are annular, *i.e.*, they encircle the pylorus, and so cause stricture.

Scirrhus of the Pylorus is usually a small growth, is hard, and does not ulcerate much. It does not produce abundant secondary deposits in the liver or elsewhere, but it causes a very tight pyloric stricture, and, as the patient may live for a considerable time, the stomach may become enormously dilated. The other cancers of the pylorus form larger growths, are softer, ulcerate more, and produce more secondary deposits, but, on the whole, less stricture and less dilatation of the stomach.

Cancer of the Lesser Curvature is usually encephaloid or columnar epithelioma. These also often become colloid, and are large, soft, ulcerate much, and produce many secondary deposits. By the extension of the growth across the front and back of the stomach, the viscus may become hour-glass-shaped.

Cancer of the Cardia is usually an extension of œsophageal cancer, and may even be squamous epithelioma.

Cancer of the Gastric Peritoneum occurs as a part of general peritoneal cancer. It often follows cancer of the ovary. The stomach becomes encased in a white, hard, nodular growth, which causes it to contract in all its dimensions. It may assume the shape of a large banana. Its wall is crisp, like thick indianbber, and it does not collapse when opened. The mucous membrane is thrown into folds. To the naked eye this form of cancer may resemble the form of peritonitis mentioned under Chronic Gastritis; but the microscope will show cancerous structure in one case, and simple fibrous tissue in the other.

Symptoms and Physical Signs.—If the growth be at the pylorus, it gives rise to the symptoms and physical signs of slowly developing *pyloric stricture with dilatation of the stomach* (see p. 55), but there is more pain than in simple stricture, and the vomit may be streaked with blood. At the same time, a growth may be felt in the pyloric region, especially if the patient be anæsthetized. The growth is always tender, and may be slightly movable. It is large or small, according to the nature of the growth. Cancer of the **body** of the stomach gives rise to pain and urgent vomiting almost immediately after taking food, and hæmatemesis is very common. The growth is tender, and may be large, and the part on the anterior wall of the stomach may move with respiration.

Cancer of the **cardia** presents, chiefly, œsophageal symptoms.

In all cases of cancer there is marked cachexia and emaciation, and secondary deposits may be observed, especially in the liver. The absence of hydrochloric acid from vomited material is said to indicate cancer.

Treatment.—In pyloric cancer the treatment for pyloric obstruction may be tried. In cancer of the body the passage of a tube into the stomach could hardly be tolerated. In all cases opium and gastric sedatives and rectal feeding may be necessary.

DYSPEPSIA (δύς, with difficulty ; πέπω, I digest).

By this term is meant the imperfect digestion of food in the *stomach*. Imperfect digestion from pancreatic, hepatic, or intestinal disease is not included here.

Causes.—There are two main groups of causes :

A. The food may reach the stomach in a condition unfavourable to digestion. As regards the *quality* of the food, it may be naturally difficult to digest, like cucumber or lobster, etc. ; or the patient may, through idiosyncrasy, be unable to digest some common article of diet, like beef, which others digest easily. Or the quantity may be too great. Or the food may not be properly masticated, so that starchy foods reach the stomach before they have been long enough in the mouth for the saliva to act on them, and proteid foods arrive without being properly subdivided by the teeth. An excess of fluid dilutes the gastric juice too much, while a deficiency of fluid causes the food to be too inspissated for the gastric juice to get at it. Alcohol, apart from being a fluid, stops digestion when taken in excess, but a small quantity may aid digestion by inducing slight hyperæmia of the mucous membrane.

B. The stomach may not be able to digest the food on account of (1) organic disease, and (2) functional disease.

1. **Every organic disease** (cancer, ulcer, congestion, etc.) of the stomach causes dyspepsia. Atrophy of the glands of the stomach, which is a common cause, probably, always results from chronic gastritis. In this group also must be included anæmia of the stomach, such as occurs in general anæmia. Exercise, or mental exertion during or immediately after eating, causes dyspepsia by withdrawing blood from the stomach to the muscles or brain, and thus leaving the stomach anæmic.

2. **Functional dyspepsia** may be due to (a) deficiency of pepsin ; (b) deficiency or excess of hydrochloric acid ; (c) deficiency or excess of the

muscular movements of the stomach. It must be noted that the deficiency in the gastric juice and the imperfect movements of the stomach, although often functional, must necessarily result from many organic diseases of the organ.

The expression 'nervous dyspepsia,' means that the functions of the stomach are disturbed by mental emotion, anxiety, etc. Sedentary habits cause dyspepsia by limiting the movements imparted to the stomach, and by inducing general want of tone.

Symptoms of Dyspepsia.—In what is called **Acute Dyspepsia** the food excites great pain, and the pain leads to vomiting. If such a case be *functional*, there is usually no tenderness, and the vomit does not contain much mucus. If it be *organic*, the other symptoms of gastritis, ulcer, etc., will be present.

Chronic Dyspepsia.—The symptoms are: Flatulence, with distension of the epigastrium and gaseous eructations; pyrosis; heartburn, and pain between the shoulders; *muscae volitantes* (specks before the eyes); palpitation of the heart, and other reflex phenomena; nausea and loss of appetite. The tongue is usually furred, but may be red and irritable.

Symptomatic Treatment.—If there be heartburn and acid eructations, it may be concluded that the gastric contents are too acid. In these cases alkaline mixtures, such as sod. bicarb., grs. xx., in aq. anethi, ʒi., are indicated. Ammonia is also a good alkali, and is, in addition, a stimulant.

In cases where there is much *flatulence*, rhubarb, magnesia, ammonia, peppermint-water, and other carminatives, are good. Antiseptics like creasote may be given, and charcoal biscuits may be tried.

If there is reason to believe that the gastric juice is deficiently secreted, give an alkali *before* meals, as it excites, then, the acid secretion. Bitters, like gentian, nux vomica, etc., also excite secretion. Sometimes it may be necessary to give pepsin and acid. hydrochlor. with the food, or to peptonize it.

Dinner pills containing rhubarb or podophyllin probably act best when the indigestion is in the duodenum.

In all cases of indigestion the cause must be sought for and treated. Thus, iron cures the dyspepsia of anæmic girls, and bismuth that of alcoholic men. The diet must be carefully regulated. Things inherently indigestible must be forbidden, and bad habits, like excessive tea-drinking or smoking, must be stopped. The teeth especially must be seen to.

DISEASES OF THE INTESTINES.

INFLAMMATION OF THE BOWELS— ENTERITIS.

Causes.—1. Irritation by substances which have been swallowed, such as the irritant poisons and mechanical irritants (see Acute Gastritis), improper food, or food which has left the stomach undigested. Irritation may also be caused by retained fæces, gall-stones, worms, etc. 2. **Poisons in the blood**, such as septicæmia, uræmia, etc. 3. **Specific causes.** The lesions of typhoid fever, dysentery, and tuberculosis may be said to constitute a specific enteritis. 4. **Exposure to cold.** Hot weather appears to predispose to enteritis; but the tendency to eat fruit, to take iced drinks, and to recklessly discard cloth-

ing during hot weather, probably are more prejudicial than the mere heat. 5. **Extension from an inflamed peritoneum.**

Pathological Anatomy.—Simple enteritis is often patchy—*i.e.*, here and there areas are found in which the mucous membrane is bright red, swollen, and covered with mucus, while in other places the surface is almost normal. The solitary glands are swollen, and may be ulcerated. The mesenteric glands are large, red, and soft.

Symptoms of Simple Enteritis.—There is moderate fever, with febrile urine, etc. Thirst is very marked. There is frequent griping over the abdomen, and diarrhœa, with large, watery stools, containing much thin mucus, and sometimes bile in excess, and, in bad cases, perhaps blood. There is not much abdominal tenderness unless the inflammation spreads through to the peritoneum.

Treatment.—Begin with *ol. ricini*, ʒss., to an adult. Then give astringents with opium. In mild cases bismuth is the best astringent; but in severe cases, chalk, hæmatoxylon, kino, or catechu, must be tried. Brandy often acts beneficially. The diet should consist of milk, arrowroot, ground rice, etc. Raw-meat juice may be useful.

Simple Chronic Enteritis.—This is most common in improperly-fed children, and leads to emaciation and to diarrhœa. Sometimes constipation alternates with the diarrhœa.

After death the mucous membrane is found to be pale and thickened, and secreting mucus in excess. Peyer's patches, the solitary glands, and the mesenteric glands are swollen, white, and firm.

The **Treatment** consists chiefly in regulating the diet.

DYSENTERY—ULCERATIVE COLITIS.

True dysentery is a specific disease, but it is more convenient to include it among the diseases of the intestines.

Causes.—The disease is most prevalent in tropical climates, and by the swampy banks of rivers. In fact, the districts in which it is endemic are the same in which malaria exists. It is not, however, due to the malarial poison, but to the *Amœba coli*.

This parasite is a protozoon about twice the size of a liver-cell. It is found in the stools, and when fresh its amœboid movements can easily be seen under the microscope. It has been cultivated in hay infusion, and when given to an animal it has produced ulcerative colitis, so the proof of its being the cause of dysentery is established according to Koch's law. It enters the human body in drinking water; but eating unwholesome food, alcoholism, etc., are predisposing causes.

Pathological Anatomy.—The mucous membrane of the large intestine, especially the rectum and sigmoid, becomes bright red and swollen. Then the solitary glands become distended with leucocytes, and burst, leaving round ulcers. In bad cases these ulcers spread and unite, so that large, irregular ulcers may be seen. This ulceration may cut off pieces of mucous membrane, which then look like islands of red velvet. Sometimes a fibrinous false membrane forms on the mucous membrane, or it may undergo coagulation necrosis, or slough in pieces of considerable size. Perforation is not common, because the submucous connective tissue, and that between the muscular bundles of the middle coat, increase in quantity early in the disease.

Dysentery is apt to become **chronic**, and one attack certainly predisposes to others. In old cases the ulcers may be unhealed, and the wall of the bowel may be enormously thickened by new connective tissue.

Symptoms.—At first there is ordinary diarrhœa. Then there is continual tenesmus, or desire to empty the rectum; and there is great pain both before and during defæcation. The stools consist of soft fæcal matter, mucus and blood, and the motion may be large or small. But in bad cases defæcation does not relieve the tenesmus, and pus and sloughs may be passed. If only the sigmoid and rectum be affected, the fæcal matter may consist of hard lumps in the midst of the blood and mucus. If there is much sloughing the stools are very offensive. On examination, there is tenderness over the sigmoid and colon, and even in recent cases the thickened bowel may be felt through the abdominal wall. Rectal examination causes excruciating pain. The degree of pyrexia is very variable, but exhaustion often sets in early on account of the great pain, and the patient may die in a few days. If the disease becomes **chronic** the symptoms persist, but with less intensity, and the bowel will probably be felt through the abdominal wall. Relapses during convalescence are common, and a second attack may be brought on years afterwards by indiscretions of diet, exposure, etc.

Complications.—The great one is abscess of the liver (see p. 11), but acute nephritis, pneumonia, etc., may occur.

Ulcerative Colitis.—Cases of inflammation and ulceration of the colon are met with from time to time, which do not seem to be true dysentery.

They occur in temperate climates, and in places and under circumstances not likely to breed dysentery. Also their connection with the amœba has not been established. But the symptoms and post-mortem appearances are like those of dysentery. They may lead to pyæmic abscesses in the liver, the contents of which are true pus, and not chocolate-coloured material.

Treatment. — Give ipecacuanha in large doses (grs. xxx.) with opium. Astringents, like bismuth, chalk, etc., may be useful. Locally, leeches round the anus are said to give relief; and enemata of starch or bismuth with opium, or suppositories of opium or cocaine (1 grain) are very good. The diet must be milk exclusively, and, as convalescence sets in, gruels, jellies, and broths may be cautiously given. Stimulants are usually necessary. No solid food should be allowed until the motions are solid and free from blood. In subacute or chronic cases some authorities recommend enemata of nitrate of silver.

TYPHLOITIS—PERITYPHLOITIS—APPENDICITIS.

Typhlitis means inflammation of the cæcum; perityphlitis, inflammation of the peritoneum surrounding the cæcum; and appendicitis, inflammation of the vermiform appendix.

Causes.—Foreign bodies, such as pins, cherry-stones, etc., in the appendix, may set up inflammation, which may end in perforation. Ulcers in the appendix or cæcum, or even impacted fæces may lead to the same result.

Pathological Anatomy.—As a result of the inflammation, the walls of the appendix and adjacent

cæcum become thickened, and as peritonitis usually occurs, the bowel is covered by fibrin. If perforation has occurred, a localized abscess or general purulent peritonitis may be found.

Symptoms.—The usual history is that of *constipation*, treated recklessly by aperients. *Vomiting* is common. Then there is *pain and tenderness* in the right iliac fossa, the pain occurring in paroxysms. The temperature may suddenly rise to 103° and stay high, but there is not usually any headache, as in typhoid fever. In a short time a swelling develops over the appendix, and when this extends to the cæcum a *sausage-shaped mass* can be felt filling up the iliac fossa. It is very tender, and usually resonant on percussion. It must be remembered that the swelling is thickened bowel, and not impacted fæces. The thigh may be drawn up.

In favourable cases the fever and the swelling slowly subside. If pus forms, rigors may occur, and fluctuation may be made out. The symptoms of general peritonitis (see p. 84) may supervene at any time.

One attack of appendicitis predisposes to another.

Treatment.—Absolute rest in bed, and a liquid diet, are necessary. Constipation must be treated by enemata only. Opium or belladonna may be given to stop the violent peristalsis which is causing the paroxysms of pain, and locally poultices or stupes are useful. Surgical treatment, in the shape of the evacuation of pus and the removal of the appendix, may be necessary.

TUBERCULAR ENTERITIS—TUBERCULAR ULCERATION OF THE BOWELS— TABES MESENTERICA.

Tubercles may occur in the intestines under two conditions:

1. In **Acute General Miliary Tuberculosis**, in which disease minute discrete tubercles are found all over the intestine. In this case the bacilli reach the intestine through the bloodvessels, and all the organs of the body are simultaneously affected with the growth of tubercles. As regards diagnosis, except that there may be some diarrhœa, the intestinal are quite masked by the general symptoms.

2. In **Chronic Tubercular Ulceration**.—This is the more common form, and the bacilli reach the intestine either in food (such as the milk or flesh of tubercular animals), or, as often happens in pulmonary phthisis, in sputum which has been swallowed. The tubercles first develope in a solitary gland, or Peyer's patch, and, as usual, caseate and soften. The mucous membrane over them then dies, and is disintegrated, so that an ulcer is left. By the successive formation and softening of tubercles the ulcer spreads, and it always follows the direction of the bloodvessels across the gut, so that an old tubercular ulcer has the following appearance: Its axis is transverse to that of the bowel, and it may even be quite annular, encircling the gut. Its base and edges are rough from the presence of tubercles and fibrous thickening, and where a tubercle has recently broken down, the edge of the ulcer is undermined. On the peritoneum, beneath the base of the ulcer, miliary tubercles can nearly always be seen.

Microscopically, the youngest tubercles exhibit one or more multinuclear and multipolar giant-cells, each surrounded by epithelioid cells, beyond which is a zone of small round cells. Older tubercles present areas of caseation.

Tubercular ulcers are most common in the ileum, but may occur in any part of the small or large intestine. In all cases of tubercular ulceration of the intestine, the mesenteric glands are also affected, and sometimes the glands are extremely tubercular, while the intestines are little or not at all affected. Recent tubercles appear as grayish nodules in the glands, but in old cases the whole gland becomes firm and yellow, like cheese, or soft like pus, or perhaps calcareous. The term *Tabes Mesenterica* (*i.e.*, wasting due to mesenteric disease) means tubercular disease of the intestines and mesenteric glands, and with it there is often associated *Tubercular Peritonitis*. Popularly the affection is called *Consumptive Bowels*.

Symptoms and Physical Signs of Tabes Mesenterica.—There is extreme emaciation and loss of strength. The face is pinched and the eyes sunken. The abdomen is prominent, and may be tense, but it is resonant all over. Sometimes the mesenteric glands are large enough to be felt, and if the peritoneum be affected, there may be ascites. Pain and tenderness may be present on some days and absent on others, according as the tubercles are causing much irritation or not. Diarrhœa is usually present, but may alternate with constipation. The motions may contain blood. There is usually remittent fever, but without any very high temperatures. A family history of tubercle and the presence of tubercles in other parts of the body will

aid the diagnosis. The disease may last for years, and at the end the intestine may become *lardaceous*.

Treatment.—Give ol. morrh., or syr. phosp. co., or maltine for the general health, taking care that they do not set up diarrhœa. Milk and farinaceous food are usually the best diet, but any simple food may be given unless the diarrhœa is found to be aggravated by it. As medicines, antiseptics are useful, such as iodoform ($\frac{1}{4}$ grain in a pill) or naphthalin (5 grains in a pill), etc.

ASIATIC CHOLERA.

This is a specific disease, but, like dysentery, it is most conveniently included among the diseases of the intestines.

Causes.—The disease is endemic in certain parts of India and China. In Europe it is epidemic, and the poison is conveyed by infected persons who travel from Asia to Europe, either by land across Russia, or by sea. The immediate cause is a comma-shaped bacillus, which is present in the stools, and the bacilli find their way thence into the human body in water which has been contaminated by the stools. Solid food, such as fruit which has lain on the ground, may also be contaminated by the stools, and it is possible that the dried bacilli may rise in the air and cause infection in that way. It is almost universally acknowledged that this bacillus, discovered by Koch, is the real cause of cholera, and, by cultivating it, in a particular manner, a material can be obtained which, when injected under the skin, produces a mere local inflammatory œdema, but protects the injected individual from cholera. The predisposing causes of

cholera are unwholesome food, fruit, alcoholism, etc. The comma bacillus can be killed by the acid of a healthy stomach, but if the stomach is in a state of catarrh, the bacilli may not be touched by the acid, and so reach the duodenum alive, and set up cholera.

Pathological Anatomy.—The stomach and intestines contain *rice-water* material, and their lining is sodden, pink, and velvety. The right cavities of the heart are full of blood, which is dark and thick, like tar. The skin and organs generally are shrivelled. The temperature may rise after death.

Symptoms.—These can be divided into three stages :

1. **The Stage of Evacuation.**—There is a preliminary simple diarrhoea which goes on to violent *purging*, with severe griping pains. The stools consist of a thin, whitish fluid, containing little white flocculi, and resembling the water in which rice has been boiled. Bile is quite absent. Urgent *vomiting* of the same *rice-water* material also occurs, and there is great *thirst* and *cramps* in the legs and abdomen.

2. **The Stage of Collapse.**—This may come on in a few hours. The patient is like a corpse. The eyes are sunken and the features drawn. The pulse may be imperceptible, and the respirations reduced to an occasional gasp. If a vein be opened the blood is like tar. The skin is cold, but the rectal temperature may be high. The purging and vomiting may cease, and the urine may be suppressed. Consciousness is retained, but there is complete apathy. Death usually occurs in this stage.

3. **The Stage of Reaction.**—The patient either warms up and steadily gets better, or his tempera-

ture becomes too high. In the latter case he may get into the *typhoid state*, with delirium, a dry, brown, tremulous tongue, subsultus tendinum, etc., and may die comatose, or recover with difficulty.

Occasionally the patient has a true relapse, going through all the symptoms a second time.

Some cases of cholera set in without any premonitory diarrhœa, the characteristic griping and purging being established from the first, while death in a state of collapse may occur in half an hour.

Treatment.—Some recommend castor oil, $\frac{1}{2}$ oz., frequently repeated, so as to hasten the passage of the poison through the bowel. Others recommend stimulants with carminatives, such as capsicum, ginger, nutmeg, etc. Others use astringents, like bismuth and chalk. Opium is usually given. Hot applications are used to relieve the abdominal pain and the cramps, and also for warmth in the stage of collapse. The injection of salt solution into the veins is recommended during the collapse.

The prophylaxis of cholera consists in taking sanitary precautions, and in warning individuals to avoid the predisposing causes, and never to drink unboiled water.

ENGLISH CHOLERA.

In summer, cases of severe diarrhœa and vomiting, accompanied by cramps, and going on rapidly to a stage of collapse, are often met with in England, and they may end fatally. They usually follow indiscretions of diet, such as eating green fruit, ice creams, etc. They are very like true cholera, but the stools are not usually *rice-water*, but consist

of thin mucus; and, nowadays, the absence of the comma bacillus from the stools makes the diagnosis from the Asiatic form of the disease easy. These cases may be treated like true cholera.

CONSTIPATION.

Causes.—These may be *functional* or *structural*.

The **functional** causes are: Deficiency of bile, as in obstructive jaundice and the febrile state; deficiency of the intestinal mucus and other secretions, as in the fevers, debilitating diseases, etc.; and deficiency of the muscular tone of the bowel, as in anæmia, etc. Sedentary habits and irregularity of meals also lead to constipation, and particularly the neglect to obey the regular call to empty the rectum.

The **Treatment** of functional constipation is to correct bad habits, and to order irritating food like brown bread, fruit and jam, curries and peppers, etc. A glass of cold water before breakfast is worth trying; but many cases can only be cured by the regular use of simple enemata for some months. Purgatives should be avoided, but the best are liquorice, cascara sagrada, sulphur, senna, and aloes. The aperient mineral waters, such as Friedrichshall or Hunyadi Janos may be necessary.

Constipation from structural causes means intestinal obstruction, partial or complete.

INTESTINAL OBSTRUCTION.

Causes.—1. Inside the bowel obstruction may be caused by impacted fæces, large gall-stones, which have ulcerated into the gut, enteroliths, etc.

2. In the wall of the bowel obstruction may be

caused by stricture due to cancer or the cicatricial contraction of tubercular, dysenteric, and other ulcers. Spasmodic stricture may also occur. The obstruction due to polypus, and that due to intussusception, belong also to this group.

3. **Outside the bowel** obstruction may be due to the *strangulation* of a coil of intestine by the neck of a hernial sac, or by a band of peritoneal adhesion, or by Meckel's diverticulum, or by an adherent omentum, etc. Volvulus may also be included among the causes of strangulation. The pressure of tumours may also cause obstruction, particularly in the pelvis.

Symptoms.—It is impossible here to mention all the symptoms which may occur, but cases of obstruction may be divided into those of acute obstruction and those in which the obstruction comes on slowly. To the first group belong the strangulations, volvulus, and intussusception. To the second belong the strictures (except the spasmodic), and the pressure by tumours.

Acute Obstruction.—Internal strangulation may be taken as a type. There is a sudden *pain*, usually referred to the umbilicus, and there may be vomiting and collapse. If the bowel be tightly nipped, the pain may be almost continuous. One or two motions may be passed, from the bowel below the strangulation, but after that *constipation* is absolute. There may be no flatus passed. Regular *vomiting* soon sets in, first of ordinary food, then of bile, and then of stercoraceous material. The higher the obstruction, the earlier the vomiting. After a variable time the compressed bowel becomes *tender* from peritonitis, and at this time its position may be estimated; but later the peritonitis may extend,

and the tenderness become universal. There is usually a moderate temperature and a foul tongue, while the urine may be scanty or suppressed (see p. 96). If the obstruction below down the abdomen becomes distended. If it be not released, the bowel becomes gangrenous, and death occurs from peritonitis.

Intussusception and volvulus are treated below, and spasmodic stricture with colic.

Treatment.—Opium is necessary to relieve pain, but it must be given cautiously, because by stopping the pain it may lead to the postponement of abdominal section, which is, of course, the only treatment likely to be of use.

Chronic Obstruction.—The type of this is afforded by cancer of the bowel. There is gradually increasing *constipation*, and the motions may be flattened or narrowed, and may contain blood or mucus. There is much griping abdominal pain, aggravated by taking aperients. The pain may be referred to one part of the abdomen, and in cancer a tender tumour may be felt. The obstruction may become practically absolute, and then the pain is worse, and vomiting sets in and becomes stercoraceous. Flatus is, however, often passed, and small pieces of fæces may worm themselves through the stricture. As a result of *chronic* obstruction, the bowel above the stricture becomes *hypertrophied* and *distended*, and then its peristaltic movements (now called *tormina*) can be felt and seen through the abdominal wall. The symptoms of obstruction may be temporarily relieved by the breaking down of the cancerous growth.

Death occurs from exhaustion, or from rupture of the bowel above the stricture. Secondary deposits of cancer may be found in the liver.

Treatment.—Aperients must never be given, but enemata may be tried. Nutrient enemata are necessary. Opium may be given with the same reservation as in acute cases. The surgical methods comprise colotomy, and in suitable cases the stricture may be excised, and the bowel above and below united.

For the Pathology of Cancer of the Intestine, see p. 78.

Fæcal impaction usually follows chronic constipation, but the obstruction may become complete. Enemata must be used, and, if in the rectum, the hard fæces can be removed with a scoop.

INTUSSUSCEPTION—VOLVULUS.

Intussusception consists in the invagination or enclosure of one part of the bowel within another, the intestine being, as it were, telescoped. The commonest variety is Ileo-cæcal Intussusception, in which the ileo-cæcal valve and cæcum pass into the ascending colon, the valve always remaining the lowest part of the intussusceptum, or enclosed portion of bowel. In Ileo-colic Intussusception the ileum passes through the ileo-cæcal valve.

Causes.—The immediate cause is irregular peristalsis, which may be due to irritating food, mental emotion, etc. The affection is most common in children. In adults it is nearly always due to a polypus, which, being constantly pushed down by peristalsis, eventually carries the wall of the bowel with it.

Pathological Anatomy.—An intussusception forms a sausage-shaped tumour, composed of three layers of intestinal wall. If it has occurred some time

before death, the peritoneum over it is inflamed. The intussusceptum is always congested, and bleeds readily, because its veins are compressed at its base. The lumen of the bowel is constricted, often completely so.

The **Symptoms** are those of acute obstruction, such as pain (which is usually intermittent), vomiting, the development of tenderness, etc. (see p. 73). No fæcal matter may be passed, but the passage of blood and mucus is very characteristic. Tenesmus is common. On examination a sausage-shaped tumour may be felt, usually on the right side; and there may be a hollow in the iliac fossa, due to the displacement of the cæcum up the colon. The intussusceptum may be felt per anum, or may even protrude.

If unrelieved, gangrene may occur, but the affection may become chronic. In a few cases the bowel above and the bowel below the intussusceptum become adherent to each other, in such a way that when the intussusceptum sloughs off no extravasation of fæces takes place, and the patient may recover. But even in this event stricture may afterwards occur at the ring of adhesion.

Treatment.—It is sometimes possible to reduce an intussusception by blowing air up the rectum. Unless the case be very recent, so that no necrosis has occurred, this is dangerous, and abdominal section should be performed.

Volvulus consists in the twisting of one part of the intestine round another, so as to compress the lumen. It is most common in the sigmoid flexure. The symptoms are those of acute obstruction, but there is often a most rapid development of tympanites. The treatment consists in abdominal section.

COLIC.

Colic is a painful spasmodic contraction of the muscular coat of the bowel. The same kind of contraction occurs in cases of enteritis, of intestinal obstruction, etc.; but the name **Colic** should be reserved for those cases in which there is no structural change in the wall of the intestine, and no mechanical obstruction to the onflow of its contents.

Causes.—Errors of diet which excite peristalsis without setting up inflammation; flatulence; lead-poisoning; gout; organic nervous affections, such as locomotor ataxy; and functional nervous affections, mental emotion, etc.

Symptoms.—Severe paroxysmal abdominal pain, which perhaps may lead to rigors, vomiting, and collapse. The patient feels that he would be relieved by defæcation, but no fæcal matter can be expelled. There is *no tenderness*—in fact, the pain may be relieved by pressure. In lead colic there may be a blue line on the gums, wrist-drop, or nephritis, and a history of exposure to the poison may be obtained.

Treatment.—Give a hot bath, or, at any rate, apply poultices or fomentations to the abdomen. A combination of castor-oil and opium is useful, and hot brandy-and-water may give relief.

In lead-poisoning, iodide of potash may be given in addition to the above remedies.

ULCERATION OF THE INTESTINES.

The following is a list of the ulcers which may be met with from the duodenum to the anus:

1. **Ulcers of the Solitary Glands.**—These may be met with in any part of the bowel. They are small

and round, and they occur in severe enteritis and colitis, and help to give the symptoms of those diseases.

2. The **Duodenal Ulcer** is a clean-cut excavation, and may perforate. It is like the gastric ulcer, and occurs in the part of the duodenum which is bathed by the acid gastric juice. Duodenal ulcers also occur after extensive burns and scalds.

3. **Typhoid Ulcers** are found in the lower part of the ileum. They are oval, with their long axis in that of the bowel, and are opposite to the mesenteric attachment. When recent, the edges are undermined, and the base covered by soft sloughs. Later, the base is quite smooth, and the edges not so undermined. They often perforate.

4. **Tubercular Ulcers** occur chiefly in the ileum, but may be in any part of the bowel (see p. 67). They rarely perforate, but may cause obstruction.

5. **Distension Ulcers** are most common in the cæcum. They result from the distension and inflammation which follow intestinal obstruction at a point lower down.

6. **Dysenteric or Colitic Ulcers** occur in the colon (see p. 63).

7. **Cancerous Ulcers** are most common in the rectum, sigmoid, and cæcum. Columnar epithelioma is the commonest form of cancer, and it often undergoes colloid degeneration. The growth always tends to encircle the bowel, and therefore produces stricture. When it has ulcerated, the ulcer is transverse to the axis of the bowel, and above and below it there is a projecting wall of growth. When this wall is cut into, it can be seen that the cancer is undermining the mucous membrane. Cancerous ulcers resemble a trench with the earth thrown up on each side. For Symptoms, see p. 74.

8. **Syphilitic Ulcers** are most common in the rectum. They are most irregular in outline, and when cicatrization takes place they may cause intestinal obstruction.

9. **Pressure Ulcers** result from the external pressure of strangulation or of a tumour, and may occur in any part of the bowel.

10. **Traumatic Ulcers** result from the irritation of hard fæces, etc. The anal fissure may be classed with these.

INTESTINAL WORMS—HELMINTHIASIS.

Symptoms.—Itching at the nose and anus; uneasiness in the abdomen; furred tongue and foul breath; grinding of the teeth during sleep; emaciation, notwithstanding a large appetite; convulsions, either general or local (such as movements of the eyes); the passage of the worm per anum or in the vomit.

Varieties of Worms.

1. **The Nematoda.**—These are elongated, cylindrical worms, with more or less tapering ends. They possess a mouth, alimentary canal, and anus; and they are bisexual. Some are oviparous, others viviparous.

The chief varieties of Nematode worms are (a) the *Ascaris lumbricoides*; (b) the *Oxyuris vermicularis*; (c) the *Tricocephalus dispar*; (d) the *Dochmius duodenalis*; (e) the *Trichina spiralis*; (f) the *Filaria sanguinis hominis*.

The *Filaria* is at no time an intestinal worm, but it causes chyluria (see p. 127).

(a) The *Ascaris lumbricoides*, or Round Worm, inhabits the small intestines. It is about eight or

nine inches long, and has three papillæ at the head. It possesses the characteristics of the other Nematoda, and is oviparous. It reaches the intestine in vegetables or water. It is best treated with san-tonin (5 grains), followed by an aperient.

(b) The *Oxyuris vermicularis*, or Thread Worm, inhabits the rectum, and may reach the vagina. It is less than half an inch long, and like a piece of thread. It is a typical Nematode worm, and is oviparous. It reaches the intestine in dirty food, or by sucking dirty fingers. It is best treated by enemata of infusion of quassia. Aperients are also useful.

(c) The *Tricocephalus dispar*, or Whip Worm, is so called because it has a hair-like or whip-like prolongation from the head. It lives in the cæcum, and is about an inch long. It is oviparous.

(d) The *Dochmius*, or *Anchylostomum duodenale*, lives in the duodenum, and by sucking the blood produces anæmia. It is a little larger than a thread-worm.

(e) The *Trichina spiralis* is a Nematode worm, but it is viviparous, and it requires two hosts. It lives, perhaps for years, in the muscles of the pig and other animals. During this time its sexual organs are not developed, and it lies coiled up and surrounded by a capsule. When the muscles of the pig are eaten in an improperly-cooked condition by man, the worms develop sexual organs and breed at once. As they pass down the intestines young ones are produced, which burrow through the intestinal wall, reach the muscles, and there become encapsuled and quiescent. The old worms which came from the pig die, and do not get to the muscles.

Trichinous pork is, to the naked eye, speckled with grayish-white dots.

Symptoms of Trichinosis.—There are two stages. First there is abdominal pain and tenderness, with diarrhœa and fever for a few days. At this point the disease may resemble typhoid fever. But when the embryos reach the muscles, these become hard, swollen, and very tender and painful. The fever may become worse, and the patient may die. The muscular symptoms distinguish the disease from typhoid, and if a piece of muscle be removed by a small harpoon, made for the purpose, the worm may be seen under the microscope. Even the adult worm is very minute.

The **Treatment** consists in treating symptoms.

2. **The Cestoda, or Tape-worms.**—All mature tape-worms possess a head and a series of flat segments called proglottides. Each proglottis contains a complete set of male and female generative organs (so that the worm is unisexual), and along their sides run the canals of a water-vascular system, by means of which the whole organism is nourished. On one or other edge of each proglottis is an opening, which leads to the generative organs. The proglottides get longer and more mature the further they are from the head. They may be three-quarters of an inch long. The great characteristic of the tape-worms is that they have two stages of existence, and require a different host for each stage. In one stage they are tape-worms, and inhabit the intestine, and in the other they are cystic organisms, and live in the muscles, liver, etc. All the Cestoda are oviparous.

The chief varieties of tape-worms are: (a) the *Tænia solium*; (b) the *Tænia mediocanellata*; (c)

the *Bothriocephalus latus*; (d) the *Tænia echinococcus*.

The *Echinococcus* is an intestinal tape-worm in the dog, and a cystic organism in the liver, etc., of man and sheep (see p. 17). The other three are intestinal tape-worms in man, and cystic in other animals; but the *Tænia solium* may also live its cystic stage in the brain, etc., of man.

(a) The *Tænia solium* has a head containing four suckers, and a proboscis or rostellum, with about twenty-six hooklets arranged in a ring. If the ripe ova, liberated from the proglottides, are passed per anum and swallowed by the pig, an embryo is hatched in the pig's stomach, and then bores its way to the muscles, where it grows into a small cyst, called the *Cysticercus cellulosæ*. Then, if the pig's flesh be eaten, improperly cooked, by man, the head of the original embryo hangs on to the intestinal wall by its suckers, the cyst drops off, and the proglottides develop.

(b) The *Tænia mediocanellata* has a cubical head with four suckers, no proboscis, and no hooklets. It passes its cystic stage of existence in the ox. It is the commonest tape-worm in England, because we like underdone beef, but are very careful about cooking pork thoroughly.

(c) The *Bothriocephalus latus* has a flat, ovoid head, with a slit on either side which acts as a sucker. It has no hooklets, and its cystic life is passed in fish, especially fresh-water fish, like the pike. It is the longest of all the tape-worms.

The treatment for tape-worm is to stop all food after four p.m., and give ʒi. of ext. filicis maris liq. in mucilage and cinnamon-water, the first thing next morning. Breakfast may be taken two hours

after this dose, but at noon $\bar{3}$ ss. of castor-oil must be administered. Kamala, in $\bar{3}$ i. doses in milk or gruel, has also been recommended.

3. **The Trematoda, or Flat Worms.**—These worms have a mouth, anus, and alimentary canal, and are bisexual. They are not true intestinal worms, but may be mentioned here.

The varieties are: (*a*) the *Distoma hepaticum*, or Liver Fluke, which is common in sheep, but has been found in the gall-bladder and bile-ducts of man; (*b*) the *Bilharzia hæmatobia*, which is a worm of the urinary passages (see p. 128).

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS—INFLAMMATION OF THE PERITONEUM.

Causes.—1. **Perforation** of the stomach; of the intestines, as in typhoid or appendicitis; of the gall-bladder, by gall-stones, etc. The rupture of hepatic and other abscesses, and the openings made by wounds and operations, also belong to this group. 2. **Extension of Acute Inflammation** from the uterus, from the intestines, as in enteritis, etc. 3. **Poisons in the Blood**, as in septicæmia, uræmia, etc. 4. **Cold.** 5. **Injury** apart from perforation.

Pathological Anatomy.—The surface of the peritoneum is red, and has lost its polish. It is usually covered by lymph (*i.e.*, fibrin mixed with leucocytes), which glues the coils of the intestine to each other and to the other viscera. The intestines are dis-

tended. In some cases there is a fair amount of turbid serum present, and in many cases pus.

If the patient recovers, fibrous adhesions may form between the abdominal viscera by the growth of granulation tissue from the subendothelial connective tissue.

Symptoms.—The onset is marked by pain in the abdomen, vomiting, and a rigor, followed by pyrexia. If perforation be the cause, collapse may precede the rigor. The pulse is quick, small, and *wiry*. The breathing is frequent and *thoracic*, because the movements of the diaphragm cause pain, and the legs are drawn up so as to relax the abdominal muscles. The tongue is dry, small, and glazed, and may become brown. There is constipation from paralysis of the bowel. The urine may be almost suppressed. The mind remains clear, and the expression is anxious.

The abdomen at first may be rigid, but soon becomes tympanitic from the distension by gas of the paralyzed intestines. It is resonant all over, very painful, and very tender.

If the patient recover, the adhesions may lead to intestinal obstruction, chronic abdominal pain, etc.

Treatment.—A cradle should be placed over the abdomen to keep the bedclothes from touching it. Fomentations or leeches may be applied. Opium must be given to stop the pain, and enemata to open the bowels. Cases occasionally occur in which it is worth while to open the abdomen and wash out the peritoneum.

CHRONIC PERITONITIS.

This may be (1) simple or (2) tubercular.

1. By **Simple Chronic Peritonitis** is usually meant old peritonitis, with adhesions, following an acute attack. But there is a rare affection called Peritonitis Deformans, which for the present we may consider a true chronic peritonitis. It is characterized by an enormous fibrous thickening of the subendothelial connective tissue of the peritoneum, which distorts all the organs, particularly the stomach (see p. 51), and by shortening the mesentery causes the intestines to be bunched together. It may convert the omentum into a transverse bar across the epigastrium. During life it is usually taken for cancer, because it leads to ascites and to the formation of nodules within the abdomen, and even after death it may only be distinguishable from cancer by the microscope.

2. **Chronic Tubercular Peritonitis** is most common in children, but may occur in adults. It may co-exist with tubercle elsewhere, more particularly with tuberculosis of the intestines and mesenteric glands (see p. 67).

Pathological Anatomy.—Two conditions may be found in this disease. In one there is ascites, and the surface of the peritoneum is covered with tubercles. In the other (which is called the *adhesive* or *dry* form) the peritoneal cavity is almost completely obliterated by fibrous adhesions, among which tubercles are found, either newly formed or caseated, softened or calcified.

Symptoms.—There is progressive asthenia and emaciation. The abdomen is enlarged either from ascites or, in the dry form, from the distension of

the intestines with gas. Masses of tubercles may be felt through the abdominal wall, and there will probably be alternate diarrhœa and constipation. Pain and tenderness over the abdomen are present at one time and absent at another. The disease comes on very slowly, and there is usually remittent fever and night-sweats. But the patient may live for years.

The Treatment is the same as that for intestinal tuberculosis, but in many cases of the non-adhesive form the operation of opening the peritoneal cavity has been followed by apparent cure.

CANCER OF THE PERITONEUM.

This is usually secondary to cancer of the stomach, ovary, etc., but in some cases no primary cancer can be found. It is most common in women.

Pathological Anatomy.—There are nodules of cancer all over the peritoneum, and the omentum may be converted into a mass of cancer. Colloid cancer is the most common variety. Ascites is nearly always present. Sometimes the thoracic duct gets blocked, and then the lacteals of the mesentery are visible as white lines, and the fluid in the peritoneum becomes *chylous*.

Symptoms.—There is pain over the abdomen, with progressive loss of strength and emaciation. The abdomen becomes distended from ascites, and large tender nodules can be felt through the abdominal wall, especially over the omentum. The fluid withdrawn by paracentesis is often sanguineous, and sometimes chylous. The growths increase rapidly, and death occurs in a few months.

ASCITES.

Ascites means the accumulation of serum in the peritoneal cavity.

Causes.—There are three groups of causes :

1. **Obstruction of the Portal Vein**, due to cirrhosis or cancer of the liver, enlarged glands in the portal fissure, aneurism of the hepatic artery, etc. When cardiac dropsy becomes general, ascites results from the back pressure extending through the hepatic vein to the portal vein.

2. **Hydræmia**, as in renal dropsy (see p. 113) and anæmia.

3. **Chronic Peritonitis**, due to tubercle, cancer, etc.

In the first two cases the fluid is almost pure serum ; in the last case it may deposit fibrin, and if due to cancer it may contain blood or chyle. Ascitic fluid is always highly albuminous.

Symptoms and Physical Signs.—The abdomen becomes large. In the *dorsal decubitus* the flanks bulge, and the front of the abdomen is flat. But in extreme cases the front may be prominent and the umbilicus protrude. On percussion the flanks are dull and the front resonant, because the intestines float up there. If the mesentery be short or the fluid abundant, the note in front may be dull ; but if the finger be pressed down hard, it will reach the intestine, and then percussion will yield a resonant note. If one hand be placed flat on one flank, and the other flank be flipped with the finger of the other hand, a cross-tap fluctuation can be obtained. If the patient be now put into the *lateral decubitus*, the uppermost flank becomes resonant on percussion ; but if there be many adhesions, as in chronic

peritonitis, time must be allowed for the serum to percolate through them to the dependent side.

The bulk of fluid may be so great as to compress the vena cava, causing enlargement of the abdominal veins; or the renal vein, causing scanty, high-coloured, albuminous urine: or to push up the diaphragm, causing dyspnœa. The exhaustion from the drain of albumen in the serum is very great.

Differential Diagnosis.—The other dull, fluctuating swellings of the abdomen are the ovarian cyst, the pregnant uterus, the distended bladder, the hydatid of the liver, and the hydro- or pyo-nephrosis. The first three can be distinguished from ascites by percussion, because they make the front of the abdomen dull, and leave the flanks resonant; and, further, the dulness does not alter with the position of the patient. A vaginal examination, the passage of a catheter, and other obvious considerations, will assist the diagnosis. It will be remembered that ovarian fluid is always more or less glutinous, and contains large granular corpuscles. Large hydatids of the liver also tend to come forwards and cause dulness there, while they leave the flanks resonant, and an exploring needle will withdraw a non-albuminous fluid containing hooklets. The renal tumours are one-sided, and the dulness does not change its position with that of the patient. Further, the colon is always in front of a renal tumour, and may be felt or percussed out.

Treatment of Ascites.—This depends on the cause. Portal obstruction can be treated by watery purgatives and diuretics; and renal dropsy by purgatives, diaphoretics, and iron. Painting with iodine may be tried in chronic peritonitis. As a rule, however, repeated paracentesis is necessary.

LOCALIZED ASCITES.

In this condition the ascitic fluid is pent up (in some part of the peritoneal cavity) within a sac, the boundaries of which are formed by the adhesion of the abdominal viscera to each other or to the abdominal wall. The sac may be between the coils of the intestine, between the liver and diaphragm, etc.

These cases are extremely difficult to diagnose. They give rise to a localized dullness (which does not shift with the position of the patient), and to fluctuation, and, according to their position, they may be mistaken for ovarian cysts, cholecysts, etc. Sometimes their nature can be guessed at by obtaining a history of former acute peritonitis or by the presence of other symptoms of chronic peritonitis. Again, their position may not correspond with that of the cysts which they simulate, and the other symptoms of such cysts may be absent. An exploration by needle is, however, usually necessary, and fortunately, even if abdominal section be performed, it is the best treatment in any case. In the same way an abscess may be localized in the peritoneal cavity by adhesions which form around it. But, as pus formation can usually be recognised by rigors, sweatings, and remittent fever, and as abdominal section must be performed to let out the pus, the diagnosis of the primary cause of the suppuration is less important.

ABDOMINAL TUMOURS AND SWELLINGS.

Enlargement of the abdomen may be *uniform and general*, or *local*.

1. **Uniform and General Enlargement of the Abdomen** may be due to :

(a) **Tympanites**, *i.e.*, to distension of the intestines with gas, such as occurs in acute and chronic peritonitis, in intestinal obstruction, etc. In these cases the abdomen is hyper-resonant all over.

(b) **Fluid Accumulations**, such as ascites (see p. 88).

It is hardly possible for solid tumours to cause a general and uniform enlargement of the abdomen. Malignant tumours may grow to an enormous size, but the bigger they are, the more likely they are to be nodular, and the easier they are to diagnose.

A solid tumour may be masked by ascites ; but it can often be felt by placing the tips of the fingers on the abdomen and suddenly pressing them down. Such a movement displaces the fluid, and causes the fingers to impinge on the solid mass below.

2. **Localized Abdominal Swellings** —In diagnosing these, it is necessary to remember the normal position of each abdominal viscus ; and, whether it normally moves with the respiratory movements of the diaphragm ; and, whether it normally can be moved by the hand during palpation.

It must also be borne in mind that a movable viscus may become fixed by adhesions, or may become too large and heavy to move with the diaphragm. Examples of the last are furnished by the liver in some cases of cancer, etc., and by the spleen in some cases of malaria, etc. Again, an

organ may be pushed out of its normal position (*e.g.*, the liver from tight-lacing), or it may drop out of its normal position from increase of weight (*e.g.*, the pylorus affected by cancer). Also, an organ may be congenitally displaced; for instance, the liver is occasionally found on the left side.

Lastly, it is necessary to know the diseases to which each viscus is liable, and the symptoms and physical signs afforded by those diseases.

A **Swelling in the Right Hypochondrium** may arise from the *liver*, the *gall-bladder*, the *kidney*, or the *colon*. It is only necessary to repeat here that the colon lies behind liver enlargements, and in front of kidney enlargements, and that liver enlargements, unless very large or fixed by adhesions, move with the diaphragm.

Swellings of this part of the colon may be due to cancer, or intussusception, or dysentery, or faecal impaction, or pericolic inflammation with or without abscess, the symptoms of each of which have been described.

A **Swelling in the Epigastrium** may arise from the *liver* (especially in primary cancer), the *stomach* (in dilatation or cancer), or the *pancreas*, or it may be an *aneurism*.

Pyloric tumours are often movable by the hand, while pancreatic tumours are fixed. Tumours of the left lobe of the liver and of the body of the stomach often move slightly with the respiration.

An aneurism gives rise to a bruit and thrill. Solid tumours of the epigastrium often have a pulsation communicated to them by the aorta, but this pulsation is *not expansile*.

A **Swelling in the Left Hypochondrium** may arise from the *spleen*, the *kidney*, the *colon*, the left

lobe of the *liver* (rarely), or the fundus of the *stomach* (rarely).

An enlarged spleen is always in front of the colon, in many cases the notches can be felt, and, unless very large or adherent, it moves with respiration.

A Swelling in the Umbilical Region may have originated in any of the abdominal or pelvic viscera, and have reached this region. But an *omentum*, enlarged by cancer, etc., a *localized ascites*, and an *aneurism*, may all be met with in this position.

A Swelling in the Lumbar Regions may arise from the *kidney*, or *colon*, or, if very large, from the *liver* or *spleen*. *Spinal abscesses* may also be felt in this region, in which case there should be tenderness over the *vertebræ*.

A Swelling in the Iliac Regions may arise from the *cæcum* or *sigmoid*, or be due to enlarged glands or a *spinal abscess*. Pelvic tumours may also invade this region.

A Swelling in the Hypogastrium usually arises from one or other of the pelvic viscera, including the bladder.

Such affections as cancer or tubercle of the peritoneum or abdominal lymphatic glands, localized ascites, and localized peritoneal abscess, may cause swellings in any part of the abdomen. The last two are most commonly met with in the hypogastric and iliac regions.

What is called a Phantom Abdominal Tumour is a swelling which appears definite on palpation, but disappears when the patient is put under chloroform. It is most commonly found in hysterical women, and may occur in any part of the abdomen.

DISEASES OF THE KIDNEY.

GENERAL CONSIDERATIONS.

THE examination of a patient for kidney disease comprises primarily the *examination of the urine*, and the *palpation* and *percussion* of the renal area of the abdomen. But it is also necessary to inspect the *skin* for *dropsy* and *anæmia*; the *arteries* for *high tension*; the *heart* for *hypertrophy*, and the *fundus oculi* for *retinitis*. The presence or absence of *pain* must be noted, and a look-out must be kept for *uræmic* symptoms.

Palpation.—A normal kidney may be felt in thin persons with lax abdominal walls, but, as a rule, it is only felt when it is enlarged or mobile, and, therefore, displaced. Very moderate enlargement may sometimes be detected by placing one hand beneath the last rib posteriorly and pressing forwards, while the other hand, placed in the upper and front part of the lumbar region, presses backwards. The organ may be made to play between the two hands.

Percussion.—Renal dulness is only important when the kidney is enlarged. Normally, the tympanitic resonance of the colon can be elicited at the outer edge of the erector spinæ, but as the kidney enlarges it pushes the colon forwards, and consequently there is dulness beyond the muscle, while the resonant colon note can be detected in front of

the renal tumour. In fact, the colon can often be *felt* in front of the tumour, and its presence in that position is the great test between splenic and renal tumours, even when the enlargement is so great as to fill up half the abdomen.

Anatomically, the upper limit of the kidney is at about the level of the spine of the eleventh dorsal vertebra, and the lower limit at that of the third lumbar vertebra. At a *post-mortem* the kidney should weigh about $4\frac{1}{2}$ oz., its fibrous capsule should strip off easily, leaving a smooth surface, and there should be about a quarter of an inch of cortex between the base of the pyramids and the surface of the organ.

THE PATHOLOGY OF THE URINE.

The normal composition of the urine is as follows:

						Parts.
Water...	955·0
Urea...	21·5
Uric acid	·5
Fixed salts	{	Chloride of sodium	}	16·5
		Alkaline phosphates		
		Alkaline sulphates		
		Phosphates of lime and mag- nesia...		
Extractives, <i>e.g.</i> , creatin, creatinine, pig- ments, etc.	6·5
						<hr/> 1000·0

It is said that the amount of solid matter in the urine may be roughly estimated by multiplying the last two figures of the specific gravity by 2. Thus, if the specific gravity be 1020, the amount of solids would be in the proportion of 40 parts to 1,000 parts of water.

The **quantity** of urine normally passed per diem is about 50 oz. When the quantity is distinctly greater than this, the condition is called **Polyuria**. Polyuria is not a disease, but only a symptom.

The **Causes of Polyuria** are :

The ingestion of large quantities of fluids, especially diuretic fluids like beer or tea.

Diuretic drugs.

Exposure to cold.

Diabetes mellitus and diabetes insipidus.

The early stage of the lardaceous kidney.

The late stage of the granular kidney.

Mental emotion and hysteria.

A **deficiency** in the quantity of urine passed may be due to deficient secretion, which is called *suppression*, or to *retention*. Prolonged retention, however, leads to arrest of secretion. Complete suppression of the urine is called **Anuria**.

Excluding the causes of the retention of the urine in the *bladder*, the causes of a diminution in the quantity of urine passed are :

Insufficient ingestion of fluids.

Profuse sweating.

Profuse discharges ; *e.g.*, cholera.

Pyrexia.

Passive congestion of the kidney, as in mitral disease, etc.

Acute nephritis.

The large white kidney.

The late stage of the lardaceous kidney.

The blocking of both ureters (usually by calculi).

Nervous shock, especially after operations on the bladder.

Obstruction of the upper part of the small intestine.

When, in a case of granular kidney, the urine becomes greatly diminished, there is probably an acute nephritis supervening on the chronic.

The causes of the diminution in the urine in obstruction of the small intestine are, partly the reduction of the area from which fluids may be absorbed, and partly nervous shock, which is always great in these cases, because obstruction of the small bowel is nearly always due to one of the kinds of strangulation, and strangulation leads to great bruising of the intestinal wall.

The **Colour** of the urine varies with the quantity of water it contains. In polyuria it is nearly always pale, while it is notably dark in pyrexia and in disease of the mitral valves. It is also high-coloured in pernicious anæmia (from containing an excess of the urinary pigments), while it is pale in chlorosis.

Bile and blood, of course, give their characteristic colours to the urine, and certain drugs when absorbed into the blood colour the urine, particularly *santonin*, which colours it *golden-yellow*, and *carbolic acid*, which colours it *olive-green*.

The **Specific Gravity** of normal urine varies from 1015 to 1025. All the causes of polyuria, except diabetes mellitus, and very rarely diabetes insipidus, lower the specific gravity. In some cases it may be as low as 1001. In diabetes mellitus it may reach 1050. In pregnancy, and whenever the quantity is small, the specific gravity is increased.

The **Reaction** of the urine becomes more acid during fever, and when there is an excess of uric acid. It is less acid, or even alkaline, after a full meal; and it can be made alkaline by administering acetate of potash or other alkaline salts. In debility from overwork or chronic vomiting, etc., the urine

may be *secreted* with an alkaline reaction, due to the presence of fixed alkali, while in cases of inflammation of the urinary passages with pus formation the urine becomes alkaline *after* secretion, but before it is voided. This change is due to the decomposition of urea into carbonate of ammonia through the action of bacteria. The same change in the reaction occurs when urine is exposed to the air, and it is brought about by the same agency. The degree of alkalinity may be tested by litmus or by yellow turmeric paper, which alkaline urine turns brown.

The quantity of Urea in the urine is increased in fever, and in diabetes mellitus when that disease is at its height. It is diminished in Bright's disease, and in acute yellow atrophy of the liver it may be absent. If strong nitric acid be added to urine containing an excess of urea, on cooling, white



CRYSTALS OF NITRATE OF UREA.

crystals form at the line of contact of the fluids. Under the microscope these crystals are colourless, rhomboidal plates with jagged edges.

Quantitative Tests for Urea.—There are two tests, Liebig's, and Russell and West's. The latter is the simpler, and is as follows: 15 c.c. of a standard solution of hypobromite of soda are placed in a bottle, together with a test-tube containing 5 c.c. of urine. The bottle communicates with a graduated tube which dips into water. On mixing the urine

with the hypobromite, nitrogen is given off, and enters the graduated tube, displacing the water. The tube is so graduated that each division corresponds to 0.4 grains of urea per ounce of urine; so that if twenty divisions are filled with nitrogen the urine contains 8 grains per ounce.

When estimating the quantity of urea in the urine, it is necessary to collect and mix all the specimens passed in the twenty-four hours, and to test a sample of the mixture.

URINARY DEPOSITS.

These may be crystalline or not.

Crystalline Deposits.

Uric acid.
Urates.
Oxalates.
Phosphates.
Cystine.
Leucine and tyrosine.

Non-crystalline Deposits.

Renal tube-casts.
Mucus.
Pus.
Blood.
Chyle.
Spermatozoa.
Portions of tumours.

Of the above, it must be remembered that urates and phosphates are often amorphous.

Uric Acid.—The quantity of uric acid passed is increased during fever, in gout after the attack of arthritis, in dyspepsia, and in functional affections of the liver. Urine containing excess of it is very *acid*.

It may be passed as crystals, only visible under the microscope. These are yellowish and mostly diamond-shaped; but they may be barrel-shaped, or like dumb-bells, or like very irregular stars. In other cases uric acid is passed in small particles visible to the naked eye, and called *gravel*, or *sand*. It

then looks like cayenne pepper, the red colour being due to the readiness with which it takes up the urinary pigments, for when pure it is white. Lastly,



CRYSTALS OF URIC ACID.

uric acid may occur as a definite stone, or *calculus*, and be found in the kidney or bladder. It is yellowish or reddish-brown in colour, smooth, and rather hard.

The frequency of uric acid deposits is due to the fact that the acid is very sparingly soluble.

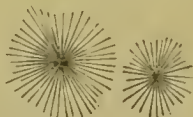
The chemical test for uric acid is the murexide test. A few grains of the acid are placed on a porcelain capsule, and covered with strong nitric acid. The fluid resulting is evaporated to dryness, and the yellowish-white powder which remains is touched with a glass rod, previously dipped in liq. ammoniæ. A red-purple colour is then produced.

Uric acid used to be called lithic acid, and urates lithates, because calculi (*λίθος*, a stone) are so frequently composed of them. Lithates must not be confused with the metal lithium.

Persons who pass uric acid or urates from time to time are said to have the *uric acid diathesis*, and the condition of blood which causes the presence of these substances in the urine is called *lithæmia*.

Urates.—The urates of soda and ammonia form the common deposit seen in the urine after taking a chill. In such cases they are amorphous. The urate of soda occurs at times in crystals, in the

shape of very fine needles arranged to form a ball ; or (especially in children) as globular crystals with spikes on the globules, the so-called hedgehog crystals. In the gouty kidney the white streaks seen



CRYSTALS OF URATE OF SODA.

in the pyramids are composed of this salt. The urate of ammonia is more rarely crystalline, but may occur as small opaque spheres or dumb-bells.

Urates are sometimes found in calculi. As a rule, however, they only form part of the stone.

Urates are deposited in, practically, the same affections as uric acid. The urine is acid, and they often acquire a pink colour from taking up pigments. They are more soluble than uric acid, and a deposit of them is quickly dissolved on boiling the urine.

Oxalates.—Crystals of oxalate of lime are often seen in the urine. They are either in the shape of regular octahedra or of dumb-bells. The former shape gave rise to the name of envelope crystals



CRYSTALS OF OXALATE OF LIME.

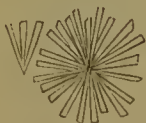
because of the four lines which meet at a point. The urine is acid. They are found in the urine in cases of dyspepsia and defective assimilation, in

functional affections of the liver, and in paroxysmal hæmoglobinuria. When present the condition is called *oxaluria*.

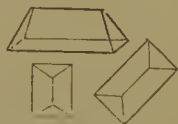
The *mulberry* calculus is composed of oxalate of lime. It is hard, dark, and rough.

Phosphates.—The white deposit which occurs on boiling faintly acid or alkaline urine consists of amorphous phosphates. Two reasons have been given for the deposit under these circumstances. One is that boiling drives off free carbonic acid from the urine, and that the absence of this acid permits the precipitation of the phosphates. The other is that boiling decomposes the urea into ammonia, and that the ammonia makes the urine sufficiently alkaline to allow the phosphates to be precipitated.

The phosphate of lime is frequently met with in the form of colourless, short needle-shaped crystals, arranged so as to form stars. It is therefore called the stellar phosphate. The ammonio-magnesian



CRYSTALS OF PHOSPHATE
OF LIME.



CRYSTALS OF TRIPLE PHOSPHATE.

phosphate (triple phosphate) also occurs as a crystal in the shape of a colourless triangular prism with bevelled ends. It has been compared to the old-fashioned glass knife-rests.

Urine containing phosphates is alkaline, and the addition of an acid dissolves the precipitate.

The affections in which phosphates are found in

the urine are (1) debility, from overwork (especially mental work), or after a severe illness, or during a chronic exhausting disease ; (2) all diseases in which pus occurs in the urine. They can also be found when the urine is made alkaline by drugs.

Phosphates form a part of many renal calculi, because the inflammation set up by a stone causes sufficient alkalinity of the urine to allow the phosphates of lime and of ammonio-magnesium to be precipitated on to the stone. The *fusible calculus* is formed largely of this deposit. Calculi of almost pure phosphate of lime, however, occur occasionally.

Cystine.—This is an organic substance containing sulphur. It has on a few occasions been found as a urinary deposit in the form of colourless six-sided plates which lie one on the other. The urine is acid, and smells of sweetbriar.



CRYSTALS OF CYSTINE.



CRYSTALS OF TYROSINE.

Calculi composed of cystine are more common. They are fawn-coloured, and are soluble in ammonia.

Leucine and Tyrosine.—In acute yellow atrophy of the liver the urine often has a greenish-yellow sediment. Under the microscope this sediment is found to be composed of crystals of tyrosine, which occur in long needles arranged like sheaves, and of leucine, which occurs in the form of fatty-looking globules.

URINARY CALCULI.

It will be seen from the above that calculi are most often composed of uric acid, oxalate of lime, or cystine, and that phosphatic calculi are usually formed by the deposit of phosphates on a calculus of another kind. Calculi of phosphate of lime, of urate of ammonia, and of carbonate of lime, are met with on rare occasions. Lastly, inspissated blood, or firmly-coagulated fibrin, may form, in the urinary passages, bodies large enough to produce the symptoms of calculi.

RENAL TUBE-CASTS.

Casts are cylindrical bodies, whose shape is due to their having occupied the renal tubules. They are common urinary deposits. The varieties of casts are as follows :

1. **Epithelial casts** contain renal epithelium, and are formed by the shedding of this epithelium. They are most common in acute nephritis and in the large white and late lardaceous kidneys.

2. **Blood casts** contain red blood corpuscles, and are due to hæmorrhage into the tubes. They are most common in acute nephritis.

3. **Hyaline casts** are quite transparent. They are due to the coagulation of some exuded material in the tubes. Chemically they are probably fibrinous ; but some think they are sometimes lardaceous. They are most common in the granular kidney, but may occur in acute nephritis.

4. **Fatty casts** contain oil globules, which are derived from fatty degeneration of the tubular

epithelium. They are most common in the large white kidney.

5. **Granular casts** are so called because they are composed of fine granules. Very finely-divided oil would present a granular appearance, but most often the granules are chemically proteid, and are derived from cloudy swelling of the renal epithelium. They occur in all forms of Bright's disease.

6. **Casts containing leucocytes** occur in acute nephritis and when suppuration is going on in the kidney.

Mixed casts, such as a hyaline cast with a leucocyte, an epithelial cell, or an oil globule in it, are, of course, constantly met with. Casts vary much in length.

MUCUS.

Mucus is often found in the urine, forming a whitish cloud, which does not quite settle to the bottom of the specimen-glass. If *liq. potassæ* be added to such urine it becomes *glairy*. The condition means an excessive secretion by the mucous glands of the bladder, which is scarcely severe enough to be called a catarrh.

PUS IN THE URINE—PYURIA.

Pus forms a white deposit in the urine. It becomes *glairy or ropy* when *liq. potassæ* is added, and under the microscope the pus corpuscles can be seen. Phosphatic crystals and bladder epithelium are often mixed with the corpuscles. Specimens of urine containing pus will give the tests for albumen, because liquor puris is an albuminous fluid ; but care must be taken to acidify, for, as has

been already stated, purulent urine is often ammoniacal.

Pus may come from the kidney, as in suppurative nephritis and tubercular kidney; or from the pelvis of the kidney, as in calculous or other forms of pyelitis and in pyonephrosis; or from the bladder, as in cystitis; or from the urethra. It is often added to the urine accidentally by the bursting of an abscess into some part of the urinary passages. Thus, the pelvic abscesses of women often burst into the bladder.

As pus from the vagina may contaminate the urine, a specimen drawn from the bladder by catheter must be examined in all cases of pyuria in women.

BLOOD IN THE URINE—HÆMATURIA.

Blood in the urine may be recognised by its colour, and by finding red blood corpuscles on microscopical examination. The corpuscles are often much smaller, and crenated by the acidity of the urine.

It sometimes happens that the hæmoglobin of the blood is found in the urine, but no corpuscles. This condition is called *Hæmoglobinuria*, and it occurs in a disease called **Paroxysmal Hæmoglobinuria**, and in certain cases of malaria.

In both hæmaturia and hæmoglobinuria the urine gives the following test for blood: Add a few drops of tincture of guaiacum to the urine, and then pour in a small quantity of ozonic ether. If blood be present, a light-blue cloud forms between the ether at the top and the urine at the bottom.

If the blood comes from the kidney or the pelvis of the kidney, it will be intimately mixed with the urine. If the quantity of blood be large, the urine

looks like porter ; if small, it has a *smoky* appearance. The distinction between blood from the kidney and blood from the pelvis of the kidney is that in the former case there are blood-casts present in the urine. If the blood comes from the bladder it is less intimately mixed with the urine, its colour is more like that of blood, and, if the quantity be small, it is usually passed at the end of micturition. Blood effused at the prostate may, however, be shot out in the shape of a small cylindrical clot at the commencement of micturition.

The Causes of Hæmaturia from the Kidney are :

1. Acute nephritis, especially when due to cold, scarlet fever, septicæmia, and poisons like cantharides or turpentine.
2. Passive congestion from disease of the mitral valve or pressure on the renal vein.
3. Sarcoma or cancer of the kidney.
4. Tuberculosis of the kidney.
5. Infarction from embolism in the kidney.
6. Injury to the kidney from wounds or blows on the back.
7. Blood conditions, such as scurvy, purpura, hæmophilia ; severe anæmias ; malignant fevers, like bad cases of typhus, small-pox, etc.
8. Vicarious menstruation.

In hæmoglobinuria the hæmoglobin also comes from the kidney.

The Causes of Hæmaturia from the Pelvis are :

1. Calculus.
2. Endemic hæmaturia, caused by the *Bilharzia hæmatobia*.
3. Sarcoma or cancer.
4. Injury (as above).

The Causes of Hæmaturia from the Bladder or Urethra are :

1. Very acute cystitis.
2. Calculus.
3. Tumours, such as cancer or papilloma.
4. Tubercular ulcers.
5. Endemic hæmaturia (as above).
6. Varicose veins.
7. Blood conditions (as above).
8. Vicarious menstruation.
9. Prostatitis.
10. Injury, *e.g.*, operations.
11. Gonorrhœa.

CHYLE—CHYLURIA.

Urine containing chyle has a milky appearance. On standing, a soft clot of fibrin forms at the bottom, and the top becomes creamy. Under the microscope fatty corpuscles can be found in it, and, if shaken up with *ether*, the opaque fluid becomes clear, because ether dissolves the fat. Chyluria is due to the *Filaria sanguinis hominis*.

SPERMATOOA.

Occasionally spermatozoa are present in urine. They are of no special significance.

PORTIONS OF TUMOURS IN URINE.

Minute particles of the villous tumour (papilloma) of the bladder may be found in the urine. They may be free, or covered by blood-clot. Sometimes, if the blood-clot be hardened and sections cut, the growth may be recognised under the

microscope, lying among the blood corpuscles. The characteristics of the growth are that it consists of processes which branch like a tree. This may, perhaps, be visible to the naked eye, but is very clear under the microscope. Each branch consists of a loop of bloodvessels, supported by connective tissue, and covered by several layers of rather elongated epithelial cells.

Bits of a cancer would only be recognised, in the urine, if they were sufficiently large to show, under the microscope, the fibrous stroma and alveoli of cancer, as well as the epithelial cells.

In sarcoma of the kidney, blood, with perhaps pieces of the growth, may get moulded in the ureter into cylinders, like worms, and be passed per urethram. This is, of course, more common in women.

ALBUMINURIA.

Many urinary deposits—*e.g.*, blood—are albuminous, but albumen may be present in perfectly clear urine.

Causes of Albuminuria.—

1. **Acute and Chronic Bright's Disease.**—(For the varieties and causes of Bright's disease, see p. 116.) The source of the albumen in these cases is partly the exudation of serum (an albuminous fluid) from the blood into the tubules, and partly the debris resulting from the necrosis of renal epithelium and leucocytes.
2. **Passive Congestion of the Kidneys.**—The albumen here results from the passive exudation of blood serum. It occurs in mitral disease, etc.

3. **Functional Albuminuria.**—See p. 122.

4. **Contamination Albuminuria.**—This means the admixture with the urine of albuminous fluids, like blood, pus, chyle, or semen.

These are the four main groups of causes ; but it follows that whatever leads to any of the four is a cause of albuminuria. For instance, in severe fevers, in pregnancy, in glycosuria, etc., there is often degeneration of the renal epithelium, but as there is also some inflammatory reaction, the albuminuria which results may be classed with acute tubular nephritis, *i.e.*, acute Bright's disease. In pregnancy, again, and in large abdominal tumours, albuminuria may be due to pressure on the renal vein, *i.e.*, to passive congestion of the kidney. Lastly, the albuminuria which follows exertion or an excessive diet of eggs may be considered functional.

Tests for Albumen.—(1) Boil the urine. If a white cloud forms, it is either albumen or phosphates. If the cloud is dissolved on adding nitric acid, it is phosphates. If the cloud remains after the acid is added, it is albumen. Albumen does not coagulate in alkaline urine.

(2) Hold the test-tube containing a little urine slantwise, and pour a little strong nitric acid down the side of the tube. The acid, being heavier, falls to the bottom. If a white cloud forms at the line of junction of the urine and the acid, it may be composed of albumen, or nitrate of urea, or of copaiba. If the cloud remain *after boiling*, it is albumen. If it become *crystalline* on standing, and disappear on heating, it is nitrate of urea. If it does not become crystalline, and also disappears

on heating, it is copaiba. The urine, further, would smell of copaiba.

(3) Solid picric acid, or a saturated solution of picric acid, produces a cloud when added to urine containing albumen. But peptones, mucin, and alkaloids (such as quinine, strychnine, etc.), when present in the urine, give the same reaction. It is therefore necessary to boil, as the cloud formed by these substances is dissolved by heat, while that due to albumen remains after boiling. It is said by some, however, that mucin does not altogether dissolve on heating. Picric acid can detect a smaller amount of albumen than any other reagent.

BILE IN THE URINE.

This occurs in jaundice, and the presence of bile makes the urine deep yellow, or even dark brown.

Test for Bile Pigments.—Place a few drops of urine on a porcelain plate and add strong nitric acid. If bile be present, a *play of colours* takes place.

Test for Bile Acids.—Place \bar{z} ii. of urine in a test-tube, add a small piece of loaf-sugar, and then \bar{z} ss. of strong sulphuric acid. A purple colour at the line of junction of the acid and the urine indicates the presence of bile acids. There are doubts about the reliability of this test.

SUGAR IN THE URINE—GLYCOSURIA.

Grape-sugar occurs in the urine in diabetes mellitus, in the gouty diathesis, in affections of assimilation, and in traumatic and other cerebral affections. It must be understood that, while there is

glycosuria in every case of diabetes mellitus, every case of glycosuria is not one of diabetes. In glycosuria the specific gravity of the urine is always high.

Tests for Sugar in the Urine—(1) Moore's Test.—If boiled with an equal part of liq. potassæ, saccharine urine becomes brown.

(2) **Trommer's Test.**—Add a few drops of a solution of sulphate of copper to the urine, and then add liq. potassæ until the blue precipitate first formed is redissolved. The result is a clear blue liquid, which, on boiling, yields an orange-red precipitate of suboxide of copper, if sugar be present.

(3) **Fehling's Test.**—Fehling's reagent is a sapphire blue fluid, containing in solution a mixture of salts which may be called a potassio-tartrate of copper. It must always be boiled before adding the urine, because if it does not remain clear after boiling it is not fit to use. When saccharine urine is added to Fehling's solution, and boiled, an orange-red precipitate results. This test is frequently used as a quantitative test for sugar. It is known that .05 gramme of sugar will completely decompose 10 c.c. of Fehling's solution, *i.e.*, will completely change the blue colour of the solution into the orange-red of the precipitate. Hence if 10 c.c. of Fehling be placed in the test-tube and boiled, and the urine be added drop by drop, as soon as the blue colour is all gone you know that the number of drops of urine used contained .05 gramme of sugar, and from this the number of grammes per ounce of urine can be determined. In practice it is necessary to dilute the urine to a known degree before testing.

(4) **Pavy's Test.**—This is much the same as

Fehling's test, but the reagent contains ammonia. The result of this is that the suboxide of copper is dissolved as soon as it is formed. Hence, while the reagent loses its blue colour, it does not become opaque, and so the exact moment of complete decolorization can be detected much more easily.

(5) **Johnson's Test.**—Take $\bar{5}$ i. of urine, add $\bar{3}$ ss. of liq. potassæ and \mathbb{M} xl. of a saturated solution of picric acid. If sugar be present, the urine becomes a deep claret colour on boiling. This may be used as a quantitative test as follows: A solution of acetate of iron is prepared of such a strength as to be the same colour as would result from boiling with potash and picric acid a specimen of urine containing $\frac{1}{4}$ grain of sugar per ounce. After testing the urine as above, it is diluted until its colour is the same as that of the iron solution, and it follows that, if it has been necessary to dilute it twenty times, it is twenty times as strong as the standard; *i.e.*, it contains 5 grains per ounce.

(6) **The Fermentation Test.**—Add some German yeast to the urine, and place in a warm situation. If sugar be present, it will give off bubbles of carbonic acid gas, and alcohol will be found in the remaining fluid. This can be used as a quantitative test, for after fermentation the urine has a diminished specific gravity, and it has been found that the loss of each degree of specific gravity corresponds to 1 grain of sugar per ounce. So that if the urine had a specific gravity of 1050 before fermentation, and of 1005 afterwards, it contained originally 45 grains of sugar per ounce. The disadvantage of this test is that it takes more than twenty-four hours.

RENAL DROPSY.

An exudation of serum among the subcutaneous connective-tissue fibres, and into serous cavities, occurs in certain forms of kidney disease, and constitutes renal dropsy. Whereas cardiac dropsy always begins in the feet, renal dropsy may begin in the eyelids, scrotum and prepuce, and in all parts where the connective tissue is loose. There may, eventually, be dropsy of all parts.

Renal dropsy is due chiefly to a deterioration of the walls of the capillaries, which allows the serum to leak through. This deterioration is, in turn, due to a deficiency of albumen in the blood, on account of the loss of albumen by the kidney. Another factor in producing renal dropsy is the retention of water in the body, on account of its non-elimination by the kidney. As a matter of fact, in practice, we find that renal dropsy is most likely to occur when the urine is scanty and contains much albumen. This is the case in acute nephritis, the large white, and the late lardaceous kidney.

The pallor produced by kidney disease is partly due to the dropsy, and partly to anæmia, the red blood corpuscles being deficient in number.

CARDIO-VASCULAR CHANGES IN KIDNEY DISEASE.

These consist in *hypertrophy of the heart*, and *increased arterial tension*, with *thickening of the arterial walls*.

The *hypertrophy of the heart* is due to the increased arterial tension, which impedes the onflow

of blood, and therefore necessitates increased heart's action.

The *increased arterial tension* is due, in acute nephritis, to spasm of the muscular coat of the arteries. This spasm is attributed to the passage, through the artery, of blood which contains a poison which the kidney fails to eliminate.

When acute nephritis becomes chronic, and in cases which are chronic from the first, the arterial wall becomes thickened. Some maintain that the thickening is a hypertrophy of the muscular coat, induced by prolonged spasm; while others say that it is an increase in the connective tissue of the inner coat—in fact, an endarteritis.

The cardio-vascular changes are most marked in the granular kidney, because that is the most chronic form of kidney disease. But they may be found in the other forms of Bright's disease, and even in other kidney diseases in which the kidney substance is nearly all destroyed, as in the cystic kidney or hydronephrosis.

In the above it has been assumed that the arterial changes follow the renal, but there are still some authorities who think that the arterial and renal changes occur together, and are part of one fibroid change throughout the body (arterio-capillary fibrosis). Such an explanation would only apply to the granular kidney.

ALBUMINURIC RETINITIS.

This is characterized by loss of vision at the centre of the field. With the ophthalmoscope white spots can be seen in the *retina*, arranged in a radiating manner round the yellow spot. There

may also be retinal hæmorrhages. In some cases there is *optic neuritis* as well as retinitis.

The retinal changes are most marked in the granular kidney.

URÆMIA.

The less grave *symptoms* of uræmia are persistent headache, with, perhaps, defects of vision; contracted pupils; attacks of asthma, or of vomiting, or of diarrhœa, or of cramps in the limbs. The grave symptoms are *convulsions* and *coma*.

Uræmic Convulsions may exactly resemble an epileptic fit, but more commonly they begin with twitching of the facial muscles, and then develop into the general convulsion. They form one variety of eclampsia.

Uræmic Coma usually comes on gradually, the unconsciousness deepening until it becomes complete. The pupils are equal, and usually contracted, the breathing stertorous, the breath foul and ammoniacal, the tongue dry and brown, and the arteries tense. There may be convulsions as well.

The cause of uræmic symptoms is a poison circulating in the blood. The poison is not urea, nor carbonate of ammonia derived from urea, but some unknown substance which is intermediate in its constitution between proteid food and urea. The defective elimination by the diseased kidney allows this substance to accumulate in the blood.

Uræmic symptoms are most common in acute nephritis and the granular kidney.

BRIGHT'S DISEASE.

By Bright's disease is meant albuminuria due to structural non-suppurative disease throughout the kidneys. The four types of Bright's disease are: 1. Acute Nephritis; 2. The Large White Kidney; 3. The Granular Kidney; 4. The Lardaceous Kidney.

Acute Nephritis.

Causes.—Scarlet fever; diphtheria; the other fevers, if severe; cold; poisons like cantharides or turpentine.

Pathological Anatomy.—The *size* of the kidney is increased, and its *capsule* peels easily. Its *cortex* is increased in size, and shows bright red points, or is mottled in red and yellow.

Microscopically.—In *scarlatinal cases* the *glomeruli* are necrotic, and crowded with necrotic leucocytes, while there are free red blood discs, leucocytes, and endothelial cells between the tuft and the capsule. Scarlatinal nephritis is, therefore, called *glomerular*. In all cases of acute nephritis the *convoluted tubes* show degenerated epithelium, the cells being fatty, or in a state of cloudy swelling. The *intertubular connective tissue* shows groups of leucocytes.

Symptoms.—Slight fever, with high arterial tension; a dull pain in the loins; pallor of the face, with œdema of the eyelids and scrotum; perhaps general dropsy. Micturition may be frequent, but the *urine* is scanty, with a high specific gravity, high-coloured, acid, and very albuminous, and it contains epithelial, granular, and small hyaline casts. In cases due to scarlatina, cold, or cantharides, the urine often contains blood, and is therefore *smoky*, and blood-casts may be found.

Mild uræmic symptoms, such as headache or vomiting, are often present; but in bad cases *convulsions* or *coma* may occur at any time, particularly if the urine become quite suppressed.

The chief complications are inflammations of serous membranes, such as pericarditis, pleurisy, etc. There may be pneumonia.

Terminations.—1. Death from uræmia or serous inflammations; 2. Complete recovery; 3. Chronic nephritis.

Treatment.—1. Free purgation, in order to eliminate the water and the poison by the bowel, and so relieve the dropsy and uræmia. Give pulv. jalap. co., $\bar{\text{ʒ}}$ i., every morning or every other morning, or pulv. scammon. co., grs. x.; or elaterium ($\frac{1}{6}$ grain) in a pill. If the patient be unconscious or convulsed, put ℥i. of croton-oil with ℥iv. of castor-oil on the tongue. 2. Free sweating, with the same object as the purging. Try hot-air baths or wet packs, or inject $\frac{1}{6}$ grain of nitrate of pilocarpin hypodermically. 3. Counter-irritation to the loins by poultices or cupping. 4. Milk diet. 5. Some recommend mild diuretics, like acetate of potash, or even digitalis, with the object of washing out the tubes. 6. As soon as the patient is improving give iron.

The Large White Kidney—Chronic Tubular Nephritis.

Causes.—Acute nephritis; alcohol, particularly beer.

Pathological Anatomy.—The *size* of the kidney is much increased; the *capsule* peels easily; the *cortex* is much increased in extent, white, or mottled in pink and buff.

Microscopically.—The *glomeruli* are normal. The *convoluted tubes* are filled with degenerated epithelial cells, which are fatty or granular. The *intertubular connective tissue* shows groups of leucocytes here and there, or little masses of young granulation tissue. This last appearance indicates the beginning of an interstitial nephritis.

Symptoms.—Great debility from the loss of albumen. Pallor and dropsy, as in acute nephritis. The *urine* is scanty, rather high-coloured, with a high specific gravity, acid, and very albuminous, and it contains epithelial, fatty, and granular casts. The appearance of blood would mean the supervention of acute nephritis.

The complications are the same as those of acute nephritis, *e.g.*, pericarditis, pleurisy, etc. But pneumonia in the œdematous lungs, and erysipelas in the dropsical limbs, may occur. Only the milder uræmic symptoms, as a rule, occur; but retinitis may be present, and the cardio-vascular changes may be appreciable.

Terminations.—1. **Death** from inflammations of the dropsical parts, more rarely from uræmia; 2. **Complete recovery**, which is very rare; 3. **Chronic interstitial nephritis**.

Treatment.—The same as for acute nephritis, but iron and other tonics must be given from the first, and the amount of purgation must be regulated by that of the dropsy. It may be necessary to tap the serous cavities and puncture the legs, in order to remove the fluid. The diet may be increased to fish and vegetables, and if there be great debility meat may be tried.

A Fatty Kidney is merely a large white kidney, in which fatty degeneration of the epithelium is

very marked, and in which there are many fatty casts in the urine. There is, however, an extremely rare condition, in which the whole kidney becomes a mass of fat, much larger than the original organ.

The Granular Kidney — Chronic Interstitial Nephritis.

There are two varieties of this kidney. In both there is a growth of granulation tissue (which becomes fibrous tissue) in the intertubular connective tissue of the kidney. But in one case the growth of granulation tissue is the primary change, and when it becomes fibrous tissue and contracts, the kidney becomes a *small granular kidney*. In the other case, the growth of granulation tissue occurs, as a secondary change, in a kidney which is much enlarged from tubular nephritis. Consequently, even when the fibrous tissue is formed and contracts, the kidney never gets much smaller than normal, and is called the *large granular kidney*.

The Small Granular Kidney.

Causes.—Gout (it is often called the gouty kidney); alcohol, especially spirits; syphilis; malaria; lead-poisoning.

Pathological Anatomy.—The kidney is very small and hard; its *capsule* peels with difficulty, tearing the kidney substance, and leaving a granular surface; its *cortex* is much diminished in thickness. In gout the *pyramids* may be streaked with urate of soda. In addition, *cysts* filled with clear or colloid fluid may be seen under the capsule, or in the cortex. *Microscopically* the *glomeruli* may be

distended to form cysts, or may show a thick fibrous capsule, or may be converted into a hyaline nodule. The *convoluted tubes* are compressed in some places and dilated in others. Where dilated, their epithelium may have accumulated, or may have fallen out. They often contain hyaline casts. The *intertubular connective tissue* is occupied by *granulation tissue* in various stages of development; i.e., in some places it is young, and consists of spindle-cells and capillaries, while in others it is old, and consists of well-formed fibrous tissue. The *arteries* show a thickened intima or media.

Symptoms.—The disease is insidious, and it is either the polyuria or uræmic symptoms, such as cramps or persistent headache, which first attract attention. Sometimes loss of vision from retinitis is the first symptom. The *urine* is copious, acid, pale, with a low specific gravity, and contains only a little albumen, with large granular and hyaline casts. There is little or no dropsy. The cardiac hypertrophy and arterial thickening are very marked; and vomiting, diarrhœa, asthma, convulsions, and coma may occur. There is emaciation, as a rule; and subcutaneous hæmorrhages, or epistaxis, hæmatemesis, melæna, or metrorrhagia may occur.

The Large Granular Kidney.

Causes.—It always follows chronic tubular nephritis.

Pathological Anatomy.—The appearances are the same as those of the small granular kidney, except that (1) the organ is about normal size, or a little large; (2) the *cortex* is about normal size, and is mottled in pink and yellow; (3) microscopically the

convoluted tubes are filled with degenerated epithelium.

Symptoms.—The indications that a large white kidney is contracting are that the urine becomes more abundant and less albuminous. The dropsy clears up, but uræmic symptoms and the cardiovascular changes become more marked, and retinitis is more likely to occur.

Complications of the Granular Kidney (either variety).—Inflammation of serous membranes; chronic endocarditis sometimes leading to aortic valvular disease; and cerebral hæmorrhage, leading to hemiplegia. The last is brought about by the high arterial tension, the brittle arterial wall, and the powerful heart's impulse, all of which tend to produce rupture.

Terminations.—Death from uræmia, cerebral hæmorrhage, or other complications. Patients may, however, be kept alive for years.

Treatment.—Some do best on an exclusively milk diet, others require fish or meat. As in all kidney affections, alcohol must be avoided, claret being the best wine to order, if any. Cold must be carefully avoided, and systematic purging and sweating may be necessary to keep off uræmia. Iron and other tonics must be given.

The Lardaceous Kidney.

Causes.—Prolonged suppuration from any cause; syphilis.

Pathological Anatomy.—The kidney is large; the *capsule* peels easily; and the *cortex* is increased in size, pale, and glistening. On applying iodine, dark-brown specks or streaks may be seen. *Micro-*

scopically lardacein may be seen in the arterioles of the *glomeruli*, and in the walls of the other arteries. When stained with methyl violet, the lardacein becomes red, while the rest of the section is blue. Old lardaceous kidneys usually exhibit chronic tubular nephritis, and sometimes even become granular. Conversely, many large white kidneys show lardaceous particles in the arterioles.

Symptoms.—The urine is at first very abundant, and contains only a little albumen; but as time goes on it becomes less abundant, and contains more albumen. This change, together with a history of suppuration and symptoms of lardaceous disease in other organs, establishes the diagnosis. With the increase of albumen dropsy may set in, and eventually there may be uræmia.

Treatment.—Cure the suppuration if possible, and attend to the general health.

N.B.—It is worthy of note that an increase in the quantity of the urine plus a diminution in the albumen indicates the contraction of a large white kidney, while a decrease in the quantity of urine plus an increase in the albumen indicates a lardaceous kidney.

FUNCTIONAL ALBUMINURIA.

By functional albuminuria is meant the presence of albumen in the urine (when secreted) without any structural disease of the kidneys. Its characteristics are that it is *intermittent*, being sometimes present and sometimes absent. The intermission may be *cyclical*, *i.e.*, albumen may be absent in the first urine passed in the morning, present after breakfast or exercise, and absent again as the day

wears on. On the next day the same cycle may occur. It is said that such cases may be watched for years, and that they never develop dropsy, or cardio-vascular changes, or uræmia.

PAROXYSMAL HÆMOGLOBINURIA.

In this disease the patients suffer from attacks of the following character. They suddenly feel cold, shiver, and have a pain in the back; even vomiting may occur. On passing urine it is porter-coloured, and is found to contain hæmoglobin (recognised by the guaiacum test), but no blood corpuscles. There are also in it crystals of oxalate of lime and granular casts, and it will give the tests for albumen. The next specimen of urine may be quite normal. These attacks are easily brought on by cold, and may occur frequently for a few days, and then not again for months. They do not lead to structural disease, nor to death, but may at times produce anæmia.

During the attack the blood in all parts of the body contains free hæmoglobin, and it is supposed that the disease consists in a separation of the hæmoglobin from the corpuscles, probably by destruction of the latter. It is certain that the kidneys are normal, and merely let the hæmoglobin leak through. The disease may be associated with malaria.

TUMOURS OF THE KIDNEY.

True tumours must be distinguished from enlargements, such as hydro- or pyo-nephrosis. The tumours commonly met with in the kidney are sarcoma, carcinoma, tubercle, gumma, adenoma,

and fibroma. Only the first three are of clinical importance.

Sarcoma of the Kidney is most common in children. It may grow to an immense size, and may become the seat of hæmorrhage, so that large blood-cysts may be found in it. **Carcinoma** is more common in adults. These growths give rise to a tumour in the loin, having the colon in front of it. The tumour may be tender, but more often causes pain by pressure only, and it does not fluctuate unless it contain blood-cysts (which can be proved by the exploring needle). There may be blood in the urine, and casts of the ureter (composed of blood and bits of the growth) may be passed; and, lastly, the cachexia of malignant disease is present.

As regards **Treatment**, the whole kidney can often be removed.

Tuberculosis of the Kidney—Scrofulous Kidney.—Discrete, miliary tubercles may be found in the kidney in general tuberculosis, but a local tuberculosis may occur, in which large masses of tubercles form in one or both kidneys. These masses caseate, and then suppurate and soften. The **Symptoms** are, pus in the urine with bacilli, and more rarely blood; a semi-solid enlargement of the kidney, which may be felt; remittent or intermittent pyrexia; and a tubercular facies, with, perhaps, the evidence of tubercle elsewhere. The **Treatment** consists in removal.

CYSTS OF THE KIDNEY.

These comprise hydatids, cystic sarcoma, the cysts of the granular kidney, the cystic kidney, and hydronephrosis.

Hydatids may occur in the kidney, and cause a

painless fluctuating swelling in the loin. They may be diagnosed by finding hooklets, or even echinococci heads in the urine, or on aspiration. They may suppurate and cause pyrexia, or, on bursting, pyuria.

The Cystic Kidney.—In this affection both kidneys are enlarged, and converted into a mass of cysts of various sizes. The disease is probably always congenital, although the patient may live to grow up; and the cysts are supposed to be formed by some congenital obliteration of the straight tubes, and the retention of the urine higher up. The kidney may sometimes be felt during life, and uræmic symptoms may occur. The urine may contain a trace of albumen, but no casts. They can only be treated as granular kidneys.

Hydronephrosis consists in a dilatation of the pelvis of the kidney, and an absorption of the kidney substance, until the organ becomes a large bag containing clear fluid. It is due to an intermittent obstruction to the escape of urine from the ureter. It is chiefly of surgical interest.

PYELITIS AND PYONEPHROSIS—CALCULOUS PYELITIS.

By pyelitis is meant inflammation of the pelvis of the kidney, and pyonephrosis is a distension of the pelvis, as in hydronephrosis, but with a purulent instead of a clear fluid. These affections are chiefly of surgical interest, and are due, as a rule, to extension of inflammation from the bladder up the ureters. But calculous pyelitis is of great medical importance.

Calculous Pyelitis is due to the constant passage of gravel, or to definite calculi in the pelvis of the kidney.

Symptoms.—1. **Pain**: either a fixed aching in the loin, or a pain only after jolting, or *renal colic*. In renal colic there is agonizing pain in the loin, radiating widely, and shooting down to the testicle, which is retracted. There are also shivering and vomiting, and there may be hæmaturia, or a stone may be passed.

2. The **Urine** contains pus in varying quantity, and blood from time to time, particularly after jolting. Both pus and blood may only be found by microscoping the urine, but there are no casts. There may be crystals of the same kind as the stone, or the urine may become ammoniacal and deposit phosphatic crystals.

3. **Pyonephrosis** may occur, a fluctuating painful swelling forming in the loin, with the colon in front of it.

4. The most characteristic sign is the sudden disappearance of pus from the urine, accompanied by an increase in size of the tumour in the loin, and by more pain and fever. This means that one kidney only is affected, and that the stone has blocked the ureter. If the stone be dislodged, the pyuria returns in greater quantity, and the tumour, the pain, and the fever become less. Complete suppression may result from both ureters being blocked by stones.

Treatment.—If the stone be uric acid, prescribe acetate of potash and a large quantity of bland fluid. Other alkaline salts may be tried, but care must be taken that the urine does not become ammoniacal. For phosphatic calculi, dilute acids

may be given. Renal colic must be treated by a hot bath or hot applications, and by opium (if there is no reason to fear uræmia) or belladonna. Finally, the stone may be removed by operation.

ABSCESS OF THE KIDNEY.

A hydatid cyst, or a tubercular mass in the kidney, may become an abscess by suppurating. But independently of this, abscesses may be set up by pyogenic bacteria, which reach the kidney either by the urinary passages (as an extension of septic cystitis), or by the blood (as in general septicæmia). In the last case a pain in the loins, with hæmaturia or pyuria, will indicate the lesion in the kidney.

PARASITES OF THE KIDNEY.

Hydatids have been mentioned. The other common parasites are, (1) the *Filaria sanguinis hominis*, and (2) the *Bilharzia hæmatobia*.

1. **Filarial Disease—Chyluria.**—This filaria is a Nematode worm, about three inches long and very narrow. It lives in the thoracic duct or some large lymphatic vessel, and by blocking the lymph-channels it causes them to rupture and discharge their contents. When this occurs in the kidney, chyle is found in the urine (see p. 107). If the patient's blood be examined while he is at rest (*e.g.*, at night), young filariæ may be found in it, and they have also been found in the urine. The disease is most common in the tropics, and is spread by the mosquito, which sucks up blood containing the embryos, and subsequently falls into drinking-water.

2. **Endemic Hæmaturia** is due to a Trematode worm, the *Bilharzia hæmatobia*, and it is endemic in Egypt and at the Cape of Good Hope. The urine contains blood, and the ova of the parasite, which are flat and oval, with a spine at one end. They are larger than vaginal epithelium. There is great anæmia. After death the ova are found in the capillaries of all the urinary passages, and it is by rupture of these capillaries that the bleeding occurs. The parasite lives in water, and enters the body during drinking or per urethram during bathing.

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AIDS TO MEDICINE.

PART IV.

DISEASES OF THE NERVOUS SYSTEM. DISEASES
OF THE EAR. DISEASES OF THE SKIN.

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PREFACE.

ANY attempt to include the essentials of a large subject within the compass of a small book necessitates the omission of many facts which seem important to the writer, and will doubtless be missed by the reader. Nevertheless, it is hoped that there is enough matter arranged in a convenient form in this book to make it a real 'Aid' to all those who are studying medicine.

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June, 1896.

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AIDS TO MEDICINE.

DISEASES OF THE NERVOUS SYSTEM.

GENERAL CONSIDERATIONS

NERVOUS diseases are called **organic** if they are due to a lesion (such as inflammation, a hæmorrhage, etc.) of any part of the nervous system. In every organic nervous disease, it is necessary to discover (*a*) in what part of the nervous system the lesion is, and (*b*) what kind of lesion it is.

If no lesion can be found, as is the case in epilepsy, hysteria, etc., the disease is said to be **functional**. A functional nervous disease is often called a *neurosis*.

Among the functional diseases must be included those affections which result from an improper quality of the blood which circulates through the nervous system, such as the convulsions produced by anæmia of the brain, or the coma produced by the poisoned state of the blood in uræmia, diabetes, etc.

In nervous diseases it is necessary to examine the *mental functions*, the *sensory functions* (including the *special senses*), the *motor functions*, the *reflexes*, the *vesical* and *rectal functions*, and the *trophic functions*.

THE MENTAL FUNCTIONS.

Localization.—These functions are supposed to reside in the gray cortical matter of the anterior part of the brain, in front of the ascending frontal convolution.

Affections of the mental functions are chiefly of the nature of insanity (p. 56), but two conditions must be mentioned here, viz., *delirium* and *coma*.

Delirium.

In this condition the patient talks rapidly, and may not know where he is, or who he is, or to whom he is talking. He may see unreal objects, and often tries to get out of bed on some imaginary errand. In other cases associated with exhaustion, he merely mutters incoherently. In mild cases he may be roused temporarily, so as to speak and act sensibly.

Delirium is most often due to poisons circulating in the brain, such as alcohol or belladonna, or the toxins of the fevers, etc. But in some cases acute lesions, such as inflammation, may be present in the cortical gray matter.

Coma.

This means *loss of consciousness*. In some cases, such as typhus fever, delirium precedes the coma, *i.e.*, the signs of perverted mental functions are followed by the complete disappearance of those functions. When the coma comes on gradually, it may be possible, at first, to rouse the patient to a state of consciousness.

Coma may be due to injuries to the brain, such as concussion or fracture of the cranial bones, etc. The following are the common causes of coma, as met with in medical practice :

(a) Lesions of the Brain.

Hæmorrhage into the brain substance, which is called **apoplexy**. In this case the coma comes on more or less suddenly ; the *breathing* is stertorous ; the *pulse* is full, strong, and often slow ; and the *face* is flushed. The *pupils* may be unequal, and *hemiplegia* (p. 17) is present in most cases. Hemiplegia can be detected during coma by observing that the mouth droops on the paralyzed side, and that the paralyzed cheek is blown out during expiration. Also, if the arm or leg be raised off the bed and then dropped, the paralyzed limb drops in a more inert, lifeless way than the sound limb. So many cases of cerebral hæmorrhage are due to kidney disease, that albuminuria, hypertrophy of the left ventricle, and high arterial tension, are often present in apoplectic coma.

(b) Poisons in the Blood.

1. **Uræmia**. -- The unconsciousness comes on gradually, and there may be *convulsions* at any moment. The *breath* is foul and ammoniacal, and there is *high arterial tension*. The *pupils* are equal, and usually contracted, and there is no paralysis. The *urine* may be suppressed ; but if any be obtained, it is albuminous. In chronic cases the left ventricle is hypertrophied, and there may be retinitis.

2. **Diabetes.**—The coma comes on gradually, and may be preceded by giddiness, nausea, or delirium. The *breath* has a sweetish odour, and the *breathing* is quick, with deep inspirations, which are noisy but not stertorous (*i.e.*, the noise is not made at the soft palate). The *pulse* is weak, and the *pupils* are equal, and usually dilated. There is no paralysis. The *urine* contains sugar, and sometimes aceto-acetic acid. But occasionally the glycosuria disappears before the coma comes on. There may be retinitis.

3. **The Toxins of the Fevers.**—The coma comes on gradually, and is preceded by delirium. The *pulse* is quick and dicrotic. The other symptoms of the typhoid state are present, such as a dry, brown, tremulous tongue, subsultus tendinum, picking at the bedclothes, and incontinence of urine and fæces. There is no paralysis.

4. **Alcohol.**—The coma is preceded, first by excitement, and then by incoherence and inco-ordination. The breath smells of alcohol, and the *breathing* may be stertorous. The *face* is flushed, and the *pulse* is quick, full, and bounding. The *pupils* are equal, and usually dilated. There is no paralysis. If the stomach-pump be used, alcohol will be found in excess in the matter which is brought up.

5. **Opium.**—The coma comes on gradually, and for some time it is possible to rouse the patient. The *face* is pale and the skin cold, while the *pulse* is small and feeble, and the breathing slow. The *pupils* are equal, and contracted to an extreme degree. There is no paralysis, and some preparation of morphia may be found in the stomach.

The above are the common causes of coma. But, among lesions, cerebral tumour, abscess, meningitis,

etc., cause coma, together with other symptoms which will be described. There is coma also during an epileptic fit. As regards blood conditions, anæmia or congestion of the brain, and poisons like chloral and other hypnotics, produce coma.

THE SENSORY FUNCTIONS.

These include tactile sensations, painful sensations, sensations of heat and cold, and the special senses.

Localization.—Sensory impressions, which come from the skin and other parts of the body, pass through the posterior nerve-roots to the posterior gray cornua of the spinal cord. It used to be thought that they then crossed over to the opposite side of the cord, but it is now believed that they do not decussate until they reach the medulla. Tactile impressions travel up the cord in the postero-median column (band of Goll), while painful impressions travel up the gray matter round the central canal of the cord. All the sensory impressions pass through the upper parts of the medulla and pons, and then through the tegmentum of the crus cerebri. They then pass through the posterior part of the internal capsule. Some think that, after this, the impressions reach the cortical gray matter behind the ascending parietal convolution, while others think that the sensory part of the cortex is much the same as the motor part.

Hyperæsthesia means that sensation is keener than normal, so that a mere touch may cause pain. It is common in hysteria, the early stage of meningitis, etc.

Anæsthesia means loss of tactile sensation. It is found in the legs in many lesions of the spinal cord, and may occur locally when a nerve is destroyed, etc.

Analgesia means the loss of the power of feeling painful sensations. It is present in a rare disease called syringo-myelia, in which the gray matter round the central canal of the cord is infiltrated with gliomatous growth.

Hemianæsthesia means loss of sensation over a lateral half of the body. It may be *organic*, in which case the lesion is in the posterior and outer part of the internal capsule, and the special senses are not affected. It may also be *functional*, as in hysteria, in which case there is loss of vision, hearing, etc., in the eye, ear, etc., on the same side of the body as the anæsthesia.

Paræsthesia is the name given to sensations which have no real objective cause. Such are *formication*, or the feeling of ants creeping on the skin, the feeling of a belt round the waist, etc.

In locomotor ataxy, sensation is *delayed*, *i.e.*, if the patient be pricked with a pin, he does not feel it at once, but some seconds later.

Pain is common in nervous diseases.

(a) *Pain in the Head*.—This occurs in various organic diseases, particularly when the meninges are irritated, as in meningitis, or when a tumour or abscess is enlarging rapidly and causing pressure. It also occurs in functional diseases, such as *migraine*, and in blood conditions, such as anæmia, uræmia, the fevers, etc. Lastly, the pain may not be due to any cerebral affection, but to rheumatism of the scalp or neuritis of its nerves.

(b) *Pain in the back* may be due to caries of the

vertebræ or to rheumatism of the muscles. The pain in diseases of the spinal cord is mostly due to irritation of the spinal meninges and the sensory nerve-roots (both outside and inside the cord), by meningitis, tumours, etc. ; and the pain often shoots down the nerves round the trunk, or down the limbs. In acute myelitis a deep-seated pain may be produced by applying a hot sponge over the back.

(c) *Pain in the nerves* occurs in neuritis, and when tumours, etc., compress the nerve-fibres. It is often called *neuralgia*. The pain may shoot along the nerve in twinges, or there may be constant aching or burning, etc., in the part supplied by the nerve. Trophic changes (see p. 16) may accompany nerve-pains.

THE SPECIAL SENSES.

It is most important to examine the eye in nervous affections. The organs of hearing, smell, and taste are of less importance, except as regards their own affections.

The Eye.

Localization.—The centre for vision is thought to be situated in the gray cortical matter of the angular convolution.

In examining the eye in medical cases, it is necessary to note the condition of the pupil, the movements of the eyeball, the power of vision, and the appearance of the fundus when seen with the ophthalmoscope.

The *pupils* are contracted by opium and eserine, in uræmia, and in locomotor ataxy, etc. They

are dilated by atropine and other poisons. Contraction of one pupil means irritation of the third nerve or paralysis of the sympathetic (*e.g.*, aneurism at the root of the neck). Dilatation of the pupil means paralysis of the third nerve or irritation of the sympathetic.

Defects in the movements of the eyeball lead to squinting (called *strabismus*), and to double vision (called *diplopia*). Any of the oculo-motor muscles may be paralyzed (see p. 88), a condition which is called *ophthalmoplegia externa*. *Ophthalmoplegia interna* means paralysis of the muscles of accommodation and the iris, such as is so common in diphtheritic paralysis.

The *vision*, medically speaking, may be deficient from paralysis of accommodation, or from affections of the retina or optic nerve. Sometimes these defects are functional; for instance, the centre of the field of vision may be lost after exposure to a strong light, or from the toxic effects of excessive smoking (tobacco amblyopia) without any changes being visible with the ophthalmoscope.

The Ophthalmoscope.—The chief affections to be looked for with this instrument are *optic neuritis*, *atrophy of the optic nerve*, *retinitis*, and *hæmorrhages*.

The optic disc, as seen with the ophthalmoscope, is a round, pale-pink area, with bloodvessels radiating from its centre to its circumference. Its edge is sharply demarcated from the surrounding retina, which is of a deep orange-red colour. This disc is the place at which the optic nerve breaks up into its branches, and here can be seen all the changes going on in the nerve.

Optic Neuritis, or Inflammation of the Optic Nerve.
—The disc, in the earliest stage, is seen to be

reddened and swollen, both of which changes cause its edge to be less clearly demarcated from the retina. The swelling has also the effect of making the vessels on the disc tortuous. In the second stage exudation has taken place, and white patches (of fibrin and leucocytes) can be seen by the side of the vessels. The disc is now so swollen that its edge is quite blurred, and the vessels are acutely bent where they pass from the disc on to the retina. In the last stage (called the choked disc) *flame-shaped* hæmorrhages are seen on the disc from rupture of the vessels. The shape of the masses of exuded blood is due to their having to separate the radiating nerve-fibres.

The causes of optic neuritis are: tumours of all kinds and in all parts of the brain; abscesses in any part of the brain; and meningitis—suppurative, tubercular, and even chronic. In cerebral hæmorrhage, softening, etc., it is not common; but it may occur, together with retinitis, in toxic conditions, such as uræmia or diabetes. Vision, although imperfect, may be retained, unless atrophy occur.

Optic Nerve Atrophy.—This may follow neuritis, in which case it is called secondary, and may be due to any of the causes of neuritis. The disc appears white, and is very conspicuous on the red retina. But the edge may be irregular (on account of the previous swelling), and pigment (the remains of the hæmorrhages) may be seen on the disc. If the atrophy be primary, *i.e.*, not preceded by neuritis, the disc is white or grayish, with the fine red lines of the bloodvessels radiating over it. The edge is sharply defined, and the disc is small and depressed below the surface of the retina. Primary atrophy of the disc may occur in locomotor

ataxy. In both kinds of atrophy vision is completely lost.

Retinitis.—The common medical cause of retinitis is uræmia, and it is then called *albuminuric retinitis*. In it, bright white spots or patches are seen in the retina, usually arranged in a radiating manner, round the yellow spot. The patches consist of exudation, or of degenerated rods and cones. Hæmorrhages may also be seen on the retina. Vision is soon lost in the centre of the field, because the yellow spot is affected so early. If neuritis be also present, the condition is called *neuro-retinitis*.

Retinitis, closely resembling the above, may also occur in diabetes and other affections.

Retinal Hæmorrhages may occur without retinitis, as in pernicious anæmia.

Tubercles may be seen in the choroid, particularly in general tuberculosis.

Giddiness—Vertigo.

The sensation of giddiness may be felt on the occurrence of a hæmorrhage into the brain, or during the formation of a tumour or abscess. It is very common in cerebellar lesions. Giddiness is also common in ear affections, particularly in Ménière's disease, when it may be so severe that the patient cannot stand, or even sit, and vomiting may occur. Giddiness may also be due to functional affections of the liver, stomach, etc.

THE MOTOR FUNCTIONS.

Localization.—The motor area on the cortex of the brain is situated in the gray matter of the

ascending frontal and parietal convolutions, which lie on either side of the fissure of Rolando, and in the gray matter which lines that fissure. The centres for the leg occupy the highest part of this area (*i.e.*, the part nearest to the great longitudinal fissure), those for the arm the middle part, and those for the face the lowest part (near to the bifurcation of the Sylvian fissure). It may be noted here that the centre for speech (at the back part of the third left frontal convolution) is just in front of the centre for the face. Motor impulses are originated at the above centres, and then are conducted along the white fibres as follows: They pass through the corona radiata to the internal capsule. As they pass through the internal capsule, the conducting fibres to the arm lie in the middle, and are, therefore, nearest to the lenticulo-striate artery. If this artery ruptures, as it often does, the arm fibres are more damaged by the effused blood than those of the face and leg, and this is why the arm is usually most affected by an apoplectic seizure. After leaving the internal capsule, the motor fibres pass through the crista of the crus cerebri, where the deep fibres of the third nerve cross them. Then they go through the pons (where the face fibres decussate), then through the medulla. In the medulla the greater number of the motor fibres decussate, and then run down the posterior part of the lateral column of the cord (which is called the crossed pyramidal tract). A few fibres do not decussate, but run down the anterior column of the cord (in the direct pyramidal tract). The motor fibres pass successively from the pyramidal tract into the gray matter of the anterior cornu of the cord, where they break up into a network around

the big multipolar cells. From these cells the motor impulses pass along the strands which cross the antero-lateral columns of the cord, then along the anterior nerve-root to the nerves, and thence to the muscles.

The course of the motor fibres, just described, may be called the *motor path*, and a lesion in any part of it causes loss of motor power. The multipolar ganglionic cells in the anterior cornua not only preside over the motor power of the muscles, but also over their nutrition and their electrical reactions. The same cells also form part of the reflex loop (see below). Hence, if these cells are destroyed, the muscles connected with them are *paralyzed, waste rapidly, and show altered electrical reactions, and any reflex act in which they may be concerned is lost*. A lesion in any part of the motor path between the cortex and these cells causes paralysis, but no wasting or other change.

Spasm.—This is the name given to the involuntary contraction of a voluntary muscle. If the contraction be prolonged, as in tetanus, strychnia-poisoning, etc., the spasm is called *tonic*. If the contractions and relaxations of a muscle alternate rapidly, as in the second stage of epilepsy, etc., the spasm is called *clonic*. During a tonic spasm the muscles are hard and painful. The term 'spasm' is also applied to the painful contraction of involuntary muscle, *e.g.*, intestinal colic.

Paralysis.—This means the loss of power to make a muscle contract by an effort of the will. Partial loss of this power is called *paresis*.

Hemiplegia means paralysis of one lateral half of the body, certain muscles being excepted. It is an indication of brain disease, and if the brain lesion

be in the right hemisphere, the paralysis is on the left side of the body (and *vice versa*), because of the decussation of the motor fibres (see p. 11).

Paraplegia means paralysis of both lower limbs, with more or less of the adjacent trunk muscles, and often of the bladder. It is an indication of disease of the spinal cord (see p. 79).

Paralyzed muscles are usually flaccid, but may become rigid.

ELECTRICAL REACTIONS OF MUSCLES.

Faradization.—In this method a rapidly interrupted current is passed through a muscle, one electrode being placed on the back of the neck, and the other on the muscle. The effect is to cause a continuous or *tonic* contraction of that muscle. The apparatus used is an induction coil, and the strength of the current is increased by drawing out the tube. The stronger the current necessary to make the muscle contract, the weaker is the response of that muscle to faradization.

Galvanization.—In this method a continuous current of electricity is passed through a muscle. Normally, if one electrode be placed on the back of the neck, as soon as the other electrode is placed on the muscle (*i.e.*, as soon as the current is closed), a momentary contraction of the muscle occurs. Another momentary contraction occurs when the electrode is taken off the muscle (*i.e.*, when the current is opened), but no contraction occurs while the current is passing. Normally, also, the contraction which occurs when closure of the current is made by placing the cathode on the muscle is greater than that which occurs when closure is

made by placing the anode on the muscle. This is expressed shortly by saying that normally the cathodal closure contraction (C. C. C.) is greater than the anodal closure contraction (A. C. C.). The strength of the current is regulated by using a more or a less number of cells.

Muscles which are degenerating give the following reaction: *They do not contract at all to the faradic current, and they contract more strongly than normal to the galvanic current, while the anodal closure contraction becomes greater than the cathodal closure contraction.* This is called the reaction of degeneration.

THE REFLEXES.

It is unnecessary to explain the nature of a reflex act, but it must be remembered that each reflex act depends on the integrity of the *reflex loop*, and the *reflex loop* consists of the sensory nerve-fibres, the posterior nerve-root, the posterior cornu of the cord, the multipolar cells of the anterior cornu, the anterior nerve-root, the motor nerve-fibres, and the muscle concerned.

Reflexes are *lost* if any part of the reflex loop be destroyed. They are *increased* if the inhibitory influence of the brain be cut off from that part of the cord which contains the spinal portion of the reflex loop. For instance, if there be a transverse lesion of the cord, the part of the cord below this lesion is, of course, cut off from the brain, and therefore all the reflexes below the lesion are increased. On the other hand, the reflexes which belong to the part of the cord which is destroyed are lost. Thus, the reflexes often enable us to tell the position of a spinal lesion.

The best-known *superficial* reflex is the plantar reflex, but a cremasteric, gluteal, and other reflexes may be obtained in normal persons. The contraction of the pupil to light is a reflex act, but not the contraction which occurs on accommodation.

The *deep* or *tendon* reflexes comprise ankle clonus, the knee-jerk, and the wrist-jerk. Others are described. They are contractions of muscles which take place when their tendons are first passively put on the stretch, and then excited by tapping or other stimuli. They are not true reflexes, because the impulse does not travel up to the cord and back again, but merely from the tendon direct to the muscle. On the other hand, they cannot be obtained unless the reflex loop be intact. Consequently it has been suggested that the stretching of the tendon sends an impulse up to the cord and down again to the muscle along the reflex loop, and that this impulse puts the muscle into a state of excitability, so that it contracts as soon as its tendon is tapped.

Ankle clonus consists in rhythmical jerks of the foot, which occur when the foot is pressed upwards and the pressure maintained. The *wrist-jerk* is an upward movement of the hand, which is obtained by letting the hand droop and tapping the tendons at the back of the wrist. Neither of the above can be obtained in normal persons, and their presence usually indicates that there is lateral sclerosis in the spinal cord. The *knee-jerk*, or *patellar reflex*, is well known. It is increased in lateral sclerosis, and lost in locomotor ataxy, and in the neuritis caused by diphtheria, alcohol, and diabetes. The deep reflexes have been called *myotatic* contractions.

TROPHIC NERVE LESIONS.

A trophic nerve lesion is a disturbance of the nutrition of any part of the body, caused by direct nervous influence. The following are examples :

(a) The atrophy of a muscle which follows lesions of the nerve supplying it (as in neuritis), or of the multipolar cells in the anterior cornu of the cord from which that nerve springs (as in infantile paralysis).

(b) The disorganization of a joint which follows a lesion in the spinal cord (*e.g.*, Charcot's knee in locomotor ataxy).

(c) An herpetic eruption on the skin which follows irritation of the nerve supplying that piece of skin (*e.g.*, herpes zoster).

The inflammatory destruction of the eye which follows lesions of the fifth nerve is due rather to the consequent anæsthesia of the conjunctiva, which allows the accumulation of irritating particles on its surface, than to trophic action ; and the same may be said of the common bed sore, which results when the buttock is anæsthetic in paraplegia, or when it is of lowered vitality and exposed to irritating excreta in the fevers. But there is a kind of deeply destructive bed sore called the *acute decubitus*, which may occur in paraplegia (in which case it is over the sacrum) or hemiplegia (in which case it is on one buttock), and which develops so rapidly that it is considered to be due to nervous influence. Roughly speaking, it may be said that the test of an atrophy or other nutritive disturbance being trophic or not is the rapidity with which it developes. The slow atrophy of muscles from disuse is, of course, not trophic.

DISEASES OF THE BRAIN.

HEMIPLEGIA.

ALTHOUGH hemiplegia is not itself a disease, but merely a collection of paralytic symptoms resulting from a lesion in the motor area of the brain, it is so necessary to understand and remember all its phenomena, that it is best to treat it under a separate heading.

Supposing a man to have had an apoplectic fit (caused, for instance, by a hæmorrhage into his internal capsule), and to have become hemiplegic in consequence, all the symptoms of the apoplexy, such as coma, etc., may have passed off by the next day, but he will probably remain hemiplegic.

The symptoms of hemiplegia which he will then present are as follows :

He can shut both *eyes*, showing that the orbicularis palpebrarum is not paralyzed (see below, rule 1) ; but he cannot shut them as tightly as he used to be able to do, and he cannot shut the eye on the affected side and keep the other eye open. The oculo-motor muscles and pupils are normal.

The furrow by the side of the *nose* is smoothed out.

As regards the *mouth*, the affected side of the lips is smooth, and may droop a little, while the other side may be slightly raised. If told to grin and show his teeth, the affected side of the mouth

does not move; but if he should laugh naturally, both sides move (see below). On eating, the food collects between the teeth and the cheek from paralysis of the buccinator. The muscles of mastication are normal.

The *tongue* is paralyzed on the affected side, and, when protruded, the sound muscles push over the weak ones, so that the tongue points to the paralyzed side. The soft palate is normal.

The *arm* is paralyzed, the muscles nearest to the trunk being least affected; the upper arm next, the forearm next, and the hand most of all.

The *leg* is paralyzed like the arm, the foot being most affected, and the hip muscles least.

The *trunk* muscles escape. Sensation and the reflexes are normal.

The above constitutes *typical or complete hemiplegia*, and all the muscles affected (face, tongue, arm, and leg) are on the same side of the body, which is on the opposite side to the cerebral hemisphere in which the hæmorrhage took place. This complete form is characteristic of a capsular (internal capsule) lesion. If the arm and face be affected, but not the leg, or the leg and arm only, etc., the affection is called *partial hemiplegia*.

It will have been noticed that the distribution of the paralysis is irregular, some muscles on the affected side being paralyzed, while others, such as the trunk muscles, etc., escape. There are two rules which govern the distribution :

1. Bilateral muscles which always act together in performing natural movements escape. This is called Broadbent's Law. Examples are the orbiculares palpebrarum (in closing the eyes), the soft palate, the muscles of mastication and of

respiration, etc. In these cases it would appear as if one side of the brain was capable of exciting the muscles on both sides to perform these natural acts. The condition of the orbicularis palpebrarum is particularly interesting, for the natural movement of shutting both eyes is retained ; but the acquired action of shutting one eye while the other is kept open is lost. To do this action, a man must have educated one side of the brain to act without the other, and when this side is cut off by the lesion, the acquired action is lost, while the natural one is retained. The movements of the angle of the mouth come under much the same law, for the voluntary act of grinning to show the teeth cannot be performed, while the involuntary act of laughing can be executed by the affected side as well as the sound.

2. In hemiplegia the movements which are most completely lost are those which are most differentiated. For instance, the hands can perform movements with perhaps one, or one or two muscles, and these movements are of numerous kinds, and are the result of a highly specialized action of the will. The movements of the shoulder, on the other hand, are fewer, and are performed by groups of muscles that mostly act together, and, most often, the will is scarcely conscious of their action. So we may say that the hand movements are more differentiated than the shoulder movements, and we find the former more completely lost in hemiplegia than the latter. For the same reason the foot is more affected than the knee, and the knee than the hip. But the hand is more affected than the foot.

These rules are not absolute, for it sometimes

happens that bilateral muscles which are associated in their action do become affected in hemiplegia. For instance, the respiratory movements on the affected side may be less than on the sound side.

Terminations of an Apoplectic Attack with Hemiplegia.—1. Death may, of course, occur during the apoplectic fit.

2. The patient may regain consciousness (remaining hemiplegic), and may appear to be doing well. But after a day or two the temperature rises, and symptoms of the typhoid state, with a return of unconsciousness, set in, and end in death. An acute bed sore (see p. 16) may develop on the buttock of the affected side, and pneumonia is not uncommon. Such cases are due to the occurrence of great irritation, or even suppuration round the lesion in the brain.

3. The patient may recover with his life, but remain permanently hemiplegic, with or without *late rigidity*.

If *late rigidity* supervene, the paralyzed arm, forearm, and hand are stiff, and become adducted and flexed, so that the arm is drawn across the chest, and the elbow and hand are bent. The wrist-jerk also can be obtained. In the leg the toes are rigidly pointed, and, unless prevented, the knee may become flexed. Ankle clonus can be obtained, and the knee-jerk is exaggerated. All the affected muscles are rigid, but do not waste (except from disuse), and have normal electrical reactions.

Late rigidity means that the motor path, from the seat of the lesion in the brain to the anterior cornua of the cord (see p. 11), has become the seat of *sclerosis*. The multipolar cells in the anterior

cornua of the cord are not affected, and, therefore, the muscles do not waste nor lose their electrical reactions. This sclerosis is called *descending sclerosis*, because it extends from the brain downwards. It is also called *lateral sclerosis*, because the motor path affected by it is in the lateral column of the cord. It will be referred to again in diseases of the spinal cord.

4. The patient may not only live, but the hemiplegia may pass off partially. In such cases the tongue and facial muscles usually recover early, and the leg recovers before the arm. The movements of the hand being the most differentiated, and the proximity of the arm fibres in the internal capsule to the lenticulo-striate artery (which so often ruptures), account for the arm being the part which is most completely disabled. The leg may recover sufficiently to allow the patient to walk, in which case the *gait* is characteristic. In this gait, the great difficulty of the patient is to get the toe off the ground (because the foot is the most affected part of the lower limb). The next difficulty is to throw the leg forward from the knee. Now, the hip muscles being strong, the patient attains his object by drawing up the affected side of the pelvis, so as to get the foot off the ground, and by swinging the whole limb round (scraping, as a rule, the inner side of the toe along the ground, and wearing out that part of the boot), from the hip, until it gets in front of the other leg. If late rigidity has set in, there is still greater difficulty in getting the toe off the ground, and the limb cannot be swung loosely round. So the pelvis has to be greatly tilted, and the limb advanced with great effort by the hip muscles.

5. In a few cases, when the lesion has been very slight, the hemiplegia may pass off almost entirely.

Causes of Hemiplegia.—The common causes are hæmorrhage in the motor area, and embolism or thrombosis of the arteries supplying the motor area. In a few cases, cerebral tumours, or abscesses, may be situated in the motor area, and thus cause hemiplegia. Sometimes, as in hysteria or after an epileptic fit, hemiplegia is functional, *i.e.*, there is no lesion in the brain to account for it. In such cases the paralysis usually passes off in a day or two.

Diagnosis of the Causes of Hemiplegia.—In all cases of hemiplegia it is necessary to discover the nature of the lesion which has caused it, and this can best be done by studying the symptoms which accompanied the onset of the paralysis. In most cases this amounts to observing the symptoms of the apoplectic fit. The modes of onset are as follows :

1. The patient may fall as if knocked down. Coma is present ; the face is flushed ; the pupils unequal ; the pulse slow, full, and strong, and the breathing stertorous. Hemiplegia is complete, and sometimes the eyes are fixedly turned away from the paralyzed side, as if they were *looking round to see what was going on in the affected half of the brain*. This turning of the eyes is called *conjugate deviation*. Such an onset indicates that the hemiplegia is due to a large hæmorrhage which tears up the brain substance rapidly, and inflicts great injury on it.

2. The patient may have headache, giddiness, buzzing in the ears, or other slight symptoms. These increase, and eventually coma sets in, which

may be as complete and prolonged as in the first case, or may be slight and transient. Hemiplegia, however, develops. This usually indicates the rupture of a small vessel, followed by a slow escape of blood, the eventual blood-clot being of varying size.

In both the above cases the presence of albuminuria, thickened arteries, and a hypertrophied heart increase the probability of the lesion being a hæmorrhage.

3. The patient becomes suddenly hemiplegic, without much other disturbance, perhaps not even giddiness or headache. This usually indicates the impaction of an embolus in a small artery supplying the motor area, and the diagnosis will be confirmed by hearing a bruit over the heart.

If the middle cerebral artery itself, or one of its main branches, were blocked, there would probably be an attack with coma, and very likely convulsions, because nearly the whole of the motor area of the cortex (see p. 11) would be deprived of blood.

Embolism of a large artery may also cause softening of a large area of the brain, and consequent permanent mental failure, which will then coexist with the hemiplegia.

4. Thrombosis is the difficult cause to be sure of, but if the hemiplegia come on somewhat gradually, as in case 2 (but with very little disturbance, as in case 3), thrombosis might be diagnosed, particularly if the patient be in the puerperal state or have syphilis, for in these two conditions thrombosis is most likely to occur.

5. If the hemiplegia developed slowly, say in a day or two, and extended from one part to another (*e.g.*, from the face to the arm. and then to the leg), and was associated with remittent fever, headache,

vomiting, and rapidly developing optic neuritis, it would probably be due to an abscess in the brain, particularly if the patient had old ear disease or septic endocarditis.

6. If the hemiplegia developed very slowly (perhaps over a period of some weeks), and crept from one part to another, and was associated with headache, vomiting, and optic neuritis, but no fever, it would probably be due to a tumour. In many cases of tumour in the motor area the paralysis of a part is preceded by convulsive movements (Jacksonian epilepsy) in it. For instance, convulsive movements in the face, followed by paralysis of the face and convulsive movements of the arm, and then by paralysis of the arm, and so on to the leg, are very characteristic of tumour. This is partly due to the fact that tumours usually irritate a part before they destroy it, but also to the fact that tumours are most common in the gray matter of the cortex of the brain, a part which produces convulsions when irritated.

Of course hemiplegia may set in in many other ways. Sometimes the fit occurs during sleep, and the patient is surprised to find himself hemiplegic in the morning. Again, very small hæmorrhages may cause almost as little general disturbance as small emboli, and then we can only rely on the evidence of heart disease for the diagnosis. If convulsions accompany the fit which ushers in the hemiplegia, it usually means that the lesion is on the motor area of the cortex, or in the pons; but it does not help us much to determine what the nature of the lesion is, except in the case of tumours as above.

Treatment of Hemiplegia.—Once it is present, no

treatment will cure hemiplegia, but massage, passive movements, and galvanization, may help the natural tendency to restoration of power, and may keep off the rigidity which results from descending sclerosis, which is of great advantage to the patient. For the treatment of the attack, see p. 32.

THE LOCALIZATION OF CEREBRAL LESIONS.

1. Lesions in the *internal capsule* cause *complete hemiplegia*, as described above. If they affect the back part of the capsule, *hemianæsthesia* may also be present, but, as a rule, sensation soon returns.

2. Lesions in the *crus* cause *complete hemiplegia*, and, in addition, *paralysis of the third nerve on the same side as the lesion*, because the third nerve goes through the crus on its way out of the brain to the eye. Hence there is ptosis, external squint, and dilatation of the pupil (the results of paralysis of the third nerve) in the eye, on the same side as the lesion, and paralysis of the face, leg, and arm on the opposite side to the lesion. There may also be *hemianæsthesia*.

3. Lesions of the *pons* cause *hemiplegia*, which may be *crossed*, i.e., the face may be paralyzed on the same side as the lesion, because the facial fibres (not the facial nerves) decussate in the pons above the lesion, and the arm and leg on the opposite side, because they decussate in the medulla below the lesion. But a big lesion in the pons may destroy the fibres from both hemispheres, causing double complete hemiplegia, and usually killing the patient. In pons lesions the pupil is contracted, and there are often convulsions.

4. Lesions in the *medulla*, if large and sudden in

occurrence, would cause death very quickly. If they occur slowly (as is the case with tumours and sclerosis), they cause the symptoms of *bulbar paralysis* (see p. 66).

5. Lesions in the *cerebellum* cause *loss of equilibrium*, and sometimes *nystagmus*, i.e., oscillatory movements of the eyeballs. But otherwise the symptoms are very obscure. The absence of the symptoms which point to other parts of the brain may help us to a correct diagnosis by a process of exclusion.

6. Lesions in the *motor area of the cortex* (see p. 11) produce a creeping hemiplegia, preceded by convulsive movements of the part which is going to be paralyzed. A partial hemiplegia, involving the arm and leg, arm and face, or any of these three parts singly, is very characteristic of a cortical lesion. The centres for the leg, arm, and face on the cortex occupy a considerable area, and a fair-sized lesion may involve one of them without the others. But, on the other hand, in the internal capsule, crus and pons, the fibres coming from these centres are so close together that a very small lesion must almost necessarily involve all three sets of fibres.

7. Lesions of the visual (see p. 7) and auditory (the temporo-sphenoidal lobes) areas of the cortex cause loss of sight and hearing respectively.

8. Lesions of the *third frontal convolution on the left side* cause amnesia, except in left-handed people (see Aphasia).

DIAGNOSIS OF BRAIN LESIONS.

The diagnosis of brain lesions is obtained by putting together the symptoms which indicate the

nature of the lesion (see the diagnosis of the cause of Hemiplegia, p. 22), and the symptoms which indicate the position of the lesion (see Localization, p. 25). It would take too long to discuss every possible case, but the following are given as examples :

1. *Symptoms of a Hæmorrhage in the Left Crus.*—There will be hemiplegia on the right side, with ptosis, dilated pupil, and external squint in the left eye, and perhaps hemianæsthesia. This tells us that the lesion is in the crus. Then these symptoms will have developed suddenly, with, probably, coma, stertorous breathing, flushed face, and a slow, strong pulse for an hour or two. Then the arteries will be found to be rigid and tortuous, the heart hypertrophied, and the urine, probably, albuminous. Now, all these latter symptoms indicate that the lesion is a hæmorrhage.

2. *Symptoms of a Tumour in the Cerebellum.*—There will be a gradually developing loss of equilibrium, but no paralysis. This tells us that the lesion is in the cerebellum. Then there will be headache, vomiting, and optic neuritis (but no fever), all of which indicate that the lesion is a tumour. The headache is, as a rule, occipital, and there may be tenderness over that part of the head.

In the above manner the diagnosis of most simple cases can be made, and the student should now be in a position to write out for himself the symptoms of each common lesion in each common situation.

APHASIA.

This term means the loss of articulate speech, and it must be, first of all, distinguished from

aphonia, which means loss of the sounds made in the larynx. Speech, of course, consists in converting these laryngeal sounds into words by means of the muscles of the lips, tongue, and soft palate. Aphasia means the loss of this power.

There are three parts of the brain concerned in speech :

(1) The centre for the memory of words, which is situated in the third left frontal convolution ; (2) a centre for the co-ordination of the movements of speech, which is somewhere in the corpus striatum ; and (3) the parts of the brain which control the common movements of the lips, tongue, etc., and which consist of the centres for the lips, tongue, etc., in the lower part of the fissure of Rolando, the lower facial fibres going down through the crus, etc., and the nuclei of the nerves of the lips, tongue, etc., in the medulla. As each of these parts may, in rare cases, be affected singly, it follows that there are three varieties of aphasia.

1. In lesions of the third left frontal convolution, the patient cannot speak, because he does not remember the words he wishes to use. If he wishes to read a book, he cannot say so, because he has forgotten the words *read* and *book*. But in uncomplicated cases he understands what you mean when you say *read a book*, and he may be able to repeat these words after you with perfect enunciation. But he soon forgets them. He usually remembers a few words or a phrase, and says them whenever he wishes to say anything. Such a patient could not write, because he would not know the words to write.

Some authorities limit the term *aphasia* to this variety. Others call this variety *amnesia*, while

others, again, give the name of amnesia to a variety of sensory aphasia.

2. In lesions confined to the co-ordinating centre, the patient knows the words he wishes to say, and he can use the muscles of speech for other purposes; but he cannot co-ordinate these muscles so that they act together to produce the sounds he desires to make, just as in locomotor ataxy the muscles do not act together for the purpose of walking, although each muscle by itself is under proper control. This variety is called *ataxic aphasia*, and in it the patient may start a sentence correctly for a word or two, and then emit a wrong syllable or word. He knows he is wrong, and tries again, but after fresh mistakes he stops in confusion.

3. In lesions of the centres and fibres of the muscles of speech, the patient knows the words he wishes to say, and could co-ordinate the muscles, but he cannot use them either for speech or any other voluntary purpose, because they are paralyzed. This occurs in hemiplegia, in bulbar paralysis, etc., and is called *paralytic aphasia*. Many authorities do not include this variety in aphasia.

In both ataxic and paralytic aphasia the patient can write if the right arm be not paralyzed.

Lesions confined to each of the three parts mentioned above are rare, and therefore it is most common to find two or more of the above varieties of aphasia co-existing.

As the centre of speech is on the left side, it is often associated with hemiplegia on the right side of the body; but in left-handed persons the centre for speech is in the right third frontal convolution, and therefore becomes associated with left hemiplegia.

The above three varieties are called *motor*, or *efferent*, aphasia, because in all of them the patient is unable (from the causes mentioned) to send forth from his mind the impulses necessary to produce speech. But, in all, his receptive faculties may be normal, *i.e.*, he may be able to understand both the spoken and the written word. *Sensory*, or *afferent*, aphasia means that he cannot understand these, and it is divided into two varieties :

1. **Word-deafness**, in which the patient does not understand the meaning of the words which are spoken to him, although he may be sensible in other respects. Some authorities call this *amnesia*.

2. **Word-blindness**, in which the patient does not understand the meaning of a word which is written, although he may be able to pronounce it.

CEREBRAL HÆMORRHAGE—APOPLEXY.

Causes.—The three factors which constitute the predisposing cause of the rupture of a cerebral artery are: (1) brittleness of the arterial wall (due to endarteritis); (2) high arterial tension; and (3) the powerful action of a hypertrophied left ventricle. Under such conditions, any excitement or muscular effort may determine the rupture.

Chronic renal disease often leads to apoplexy, because it produces the three above factors; but syphilis, alcoholism, heavy muscular work, and all causes of chronic endarteritis, tend to produce the same result. In old people, also, the arteries are always brittle. Sometimes endarteritis causes aneurisms on the cerebral arteries, and these readily break. The vessels of gliomata are also very prone to rupture.

Post-mortem Appearances.—A small blood-clot

may be absorbed, and leave a pigmented scar, in which ruby-coloured rhomboidal crystals of hæmatoidin can be seen under the microscope.

A larger clot may be absorbed, and its place taken by straw-coloured fluid, surrounded by a fibrous capsule, thus forming a blood-cyst. A large hæmorrhage, leading to death in a short time, usually presents the following appearances after death: The convolutions at the vertex are flattened, especially on the side where the hæmorrhage began. The centrum ovale is full of red clot, which has burst into and distends the lateral and third ventricles, has filled up the iter, and reached, finally, the fourth ventricle.

Symptoms.—These depend on (1) the amount of blood effused and the rapidity with which it escapes, and on (2) the part of the brain at which the hæmorrhage occurs. The symptoms which indicate the occurrence of hæmorrhage are described under the first and second modes of onset of hemiplegia (see p. 22), and under apoplectic coma (see p. 3). The symptoms which indicate the position of the hæmorrhage are described under *Localization of Cerebral Lesions* (see p. 25). By putting these two groups of symptoms together, the diagnosis of a hæmorrhage into any particular part of the brain may be made.

Hæmorrhage *may* occur in any part of the brain, but is most common in the internal capsule, crus, and pons, and consequently hemiplegia is a very common symptom. After a large hæmorrhage the mental faculties may be impaired, especially if it affected the anterior part of the cerebral gray matter.

Treatment.—During the coma, a drop of croton-

oil may be placed on the tongue, and mustard plasters applied to the calf of the legs. Venesection is also recommended. Persons whose arteries are brittle should be warned against excitement or violent exertion.

Apoplexy means simply a stroke or knock-down blow; and as a large cerebral hæmorrhage produces this effect, the terms have become almost synonymous. But embolism or thrombosis of a large cerebral artery may produce the same sudden effect by cutting off the blood from a large part of the brain.

CEREBRAL EMBOLISM—THROMBOSIS— SOFTENING OF THE BRAIN.

The cerebral arteries are often obstructed by emboli, which consist of particles of fibrin (vegetations) which become detached from the valves of the heart, and are carried in the blood-stream until they reach an artery which is too small to let them pass.

Coagulation of blood (thrombosis) may also occur in the cerebral arteries, and occlude them. It is most often a consequence of endarteritis obliterans, due to syphilis. Thrombi may also form behind an embolus.

Pathological Effects of Embolism.—The cerebral arteries beyond the circle of Willis have bad anastomoses, or none at all; and therefore the common result of the blocking of one of them by an embolus is an *infarction*—i.e., the pyramidal-shaped piece of brain supplied by that artery dies and softens. Under the microscope the nerve-cells may be fatty, or may have disappeared, the

myelin of the white substance is broken up into droplets, and leucocytes have invaded the dead piece from the margin.

Sometimes hæmorrhage occurs into the softened brain tissue, making it a hæmorrhagic infarction, instead of a non-hæmorrhagic one. If the embolus be septic, as in ulcerative endocarditis, the infarction suppurates.

The effects of **Thrombosis** are much the same as those of embolism, but the occlusion of the artery takes place more slowly, and anastomoses may be established, so that the amount of softening may be less.

The term **Softening of the Brain** has, unfortunately, been applied to those cases of intellectual failure which occur in old people from atrophy and sclerosis of the cortical gray matter. But in such cases the brain is really hard. On the other hand, intellectual failure will, of course, follow true softening of the anterior convolutions of the brain. True softening of the brain has been divided into *white*, *red*, and *yellow* softening.

White softening usually means a non-hæmorrhagic infarction, as described above; but the term is also applied to the softened condition of the walls of the ventricles which is found in acute or chronic meningitis.

Red softening usually means a hæmorrhagic infarction, as described above; but the term is also applied to the brain when it is in a state of acute inflammation; for instance, the gray matter next to the pia mater is often acutely inflamed in acute meningitis, and it is then red and soft.

Yellow softening usually means a hæmorrhagic infarction which is undergoing decolourization; but

the term may also be applied to a piece of brain tissue infiltrated with pus.

The **Symptoms of Cerebral Embolism** depend on the part of the brain which softens in consequence of the obstruction of the artery supplying it. For instance, if the centre for speech softens, aphasia results; and if the pons softens, there is crossed paralysis, etc. (see notes on Localization, p. 25). As the left middle cerebral is the artery most frequently blocked by an embolus, and as that artery supplies the greater part of the motor area, right hemiplegia is a very common result. The indication that the lesion is an embolus is furnished by the suddenness with which the symptoms occur, and the other points mentioned on p. 23.

Intellectual failure not unfrequently follows the softening due to embolism, because the cortical centres (p. 2) are often involved.

The **Symptoms of Thrombosis** also depend on the position of the artery which is occluded. For the mode of onset, see p. 23. There is no special treatment to be recommended in these cases.

TUMOURS OF THE BRAIN.

The most characteristic brain tumour is the **Glioma**, which occurs as a pinkish gray mass, not unlike hyperæmic brain tissue. It usually has ill-defined margins, because it infiltrates the brain substance; and it often undergoes mucoid degeneration, so that collections of mucoid fluid occur in it, forming pseudo-cysts. It is also frequently the seat of hæmorrhage, because its bloodvessels are very fragile. Microscopically this tumour is really a round-celled **Sarcoma**, which grows from the

delicate connective tissue (the neuroglia) of the brain; but ganglionic cells and uerve-fibres may be found in it.

Spindle-celled **Sarcomata** may also occur in the brain. These may be encapsuled, and pseudo-cysts may be found in them.

Carcinoma is not so common, and usually grows from the dura mater. **Gummata** are fairly common, and occur mostly as caseous masses connected with the meninges. Masses of **Tubercle** large enough to be called tumours may be found not only in the meninges, but even in the brain substance.

Fibromata, **lipomata**, and other tumours, occur occasionally.

The **Cysts** which occur in the brain are mostly pseudo-cysts, derived from the degeneration of sarcomata. But sometimes no trace of the origin of the cyst can be found. Parasitic cysts, such as the *Hydatid* and the *Cysticercus cellulosæ* (the cystic stage in the life of the *Tænia solium*), are occasionally found in the brain, and give rise to the symptoms of tumour.

Symptoms.—The symptoms of cerebral tumour (irrespective of its position) are headache, vomiting, and optic neuritis. If near the surface, tenderness may be found at a point on the cranium over the tumour. The other symptoms of tumour depend on its position; for instance, if it be in the internal capsule, it will cause hemiplegia, and if it be in the crus, there will be paralysis of the third nerve as well as hemiplegia (for details, see *Localization*, p. 25). But wherever it may be, the symptoms due to the part involved come on slowly with the growth of the tumour; and if the motor tract be involved, spasmodic movements of the muscles

usually precede paralysis (see p. 24), because the tumour irritates the nerve-fibres before it destroys them.

Treatment.—Gummata may be removed by iodide of potassium and mercury. Occasionally it has been found possible to remove tumours from the brain by operation.

ABSCESS OF THE BRAIN.

Abscesses may occur in the brain in ulcerative endocarditis (by the suppuration of an infarction), or in general pyæmia. But they very often follow ear disease, and especially the ear disease which complicates scarlet fever. In these cases the sequence of events is suppuration of the middle ear, necrosis of the temporal bone, and abscess of the brain. Mostly the abscess is contiguous to the necrosed bone, but sometimes apparently healthy brain tissue intervenes. Abscesses due to ear disease are most common in the temporo-sphenoidal lobes, and in the cerebellum.

Symptoms.—The symptoms of cerebral abscess (irrespective of its position) are fever (which is remittent, but sometimes intermittent, or even relapsing), headache, vomiting, and optic neuritis. The symptoms of ear disease or heart disease may help the diagnosis. For the symptoms which depend on the position of the abscess, see notes on Localization, p. 25.

Treatment.—It is possible in a certain number of cases to trephine the skull and open and drain a cerebral abscess successfully.

CONGESTION OF THE BRAIN—ENCEPHALITIS.

Various symptoms, from simple headache or giddiness, to convulsions, delirium, and coma, have been attributed to **Congestion of the Brain**; but the exact pathology and symptomatology of this condition is too obscure to be treated of in a small book.

Encephalitis, or inflammation of the brain, apart from abscess, is very rare. It undoubtedly occurs in the parts of the brain adjacent to the inflamed meninges, or to blood-clots, etc., but in such cases its symptoms are masked by the primary lesions. Cases are, however, on record in which such brain symptoms as acute delirium, convulsions, coma, and paralysis, with fever, have been present, and after death the brain has been found to be red and soft, as if acutely inflamed, and these may be looked upon as cases of acute encephalitis.

Treatment.—In cases believed to be simple congestion, or even encephalitis, ice may be applied to the head, and drastic purgatives administered. The drugs most useful in treating the symptoms of cerebral irritation (such as convulsive movements, delirium, etc.) are bromide of potassium, chloral hydrate, and hyoscine ($\frac{1}{200}$ grain hypodermically). But it is sometimes necessary to give opium. These sedative drugs may be given whether the irritation be due to congestion, abscess, or tumour, etc.

TUBERCULAR MENINGITIS.

Post-mortem Appearances.—The convolutions at the vertex of the brain are flattened, because the ventricles are distended by fluid. They may be hyperæmic or pale from pressure. There is an

excess of fluid in the arachnoid cavity and sub-arachnoid space. The structures at the base of the brain are covered with lymph (fibrin and leucocytes). On removing this lymph, it is found that the sides of the Sylvian fissure are abnormally glued together, and that tubercles are present on the fine branches of the middle cerebral artery which lie there. Tubercles may also be seen on the branches of the anterior cerebral artery in the great longitudinal fissure, and on the posterior cerebral artery in the great transverse fissure. The brain beneath the pia mater is red and soft. The lateral and third ventricles are found distended by fluid, which is often quite clear. This gave the name of acute hydrocephalus to the disease, and the effusion is due to blocking of the venæ Galeni by inflammatory products in the pia mater. The brain around the lateral ventricles is white and soft. *Microscopically* the tubercles may be seen, forming beadlike swellings in the walls of the arterioles. The swellings consist of lymphoid cells, accumulated in the adventitia.

Symptoms.—The duration of this disease is from two to four weeks, or even longer. The symptoms may be divided into those which are nearly always early, those which may be early or late, and those which are nearly always late.

Early Symptoms.—Headache, vomiting, and constipation, the association of the vomiting with the constipation being very characteristic. Fever; a quick, irregular pulse; hyperæsthesia to light, sound, and touch; contraction of the pupils, and temporary squinting due to irritation of the nerves at the base of the brain. There may be convulsions at the outset.

Symptoms which may be Early or Late.—Optic neuritis ; retraction of the back of the neck from rigidity of the muscles there ; retraction of the abdomen ; a peculiar scream, called the *hydrocephalic cry* ; sighing respiration ; a loss of elasticity of the skin, especially of the abdomen, so that, if a fold be piuched up, it subsides slowly like the skin of an old person ; the *tâche cérébrale*, which is a vivid red blush which forms along a line made by drawing the nail across the skin ; alternate flushing and pallor, the face being at one moment scarlet and perspiring, and at auother pale and cold. The temperature may be only slightly raised, but is always very irregular. In some cases there is an apparent improvement before the final symptoms set in.

Late Symptoms.—Dilatation of the pupils (which are insensible to light), and squinting from paralysis of the muscles ; bulging of the fontanelles, if they have not closed ; rigidity (with tremors) of the limbs, the arms being often flexed and drawn forward to lie on the chest ; paralysis of the muscles of deglutition ; slowness of the pulse ; complete unconsciousness ; general convulsions. Death may be preceded by hyperpyrexia, and Cheyne Stokes respiration is ofteu observed. In this method of breathing a number of rapid respirations are taken, the rapidity becoming greater and greater up to a certain point, when the respirations more or less suddenly cease, and the patient does not breathe at all for a long period, perhaps nearly a minute. Then he starts the series of rapid respirations again.

It will be observed that the early symptoms are chiefly due to the irritative effects of the inflamma-

tion, while the late ones are due to the destructive effects, and to the compression of the brain by the effusion.

Treatment.—Nearly all cases die, but a calomel purge should be given at the start, and iodide of potash administered systematically. During the headache ice may be applied to the head, and bromides and other sedatives prescribed.

SIMPLE MENINGITIS.

Causes.—These are ear disease, general pyæmia, the infective fevers, and injury.

Post-mortem Appearances.—These are much the same as those of tubercular meningitis; but no tubercles are found, and the inflammatory exudation is often purulent, and not so often confined to the base of the brain as in the tubercular form.

Symptoms.—These also resemble the symptoms of the tubercular form, but they occur with greater rapidity, the patients dying earlier. The special eye symptoms (squinting, etc.) may be absent, unless the inflammation is very marked at the base of the brain. The absence of any signs of tuberculosis elsewhere, and of a tubercular family history, and the presence of ear disease, etc., may help the diagnosis.

The **Prognosis** and **Treatment** are the same as those of the tubercular form.

The word **Lepto-meningitis** is sometimes used for any inflammation of the pia mater.

CHRONIC HYDROCEPHALUS.

There are two chief forms of this disease—hydrocephalus externa and hydrocephalus interna. In

the former the fluid collects in the arachnoid cavity outside the brain; but as this affection is rare, it need not be described here.

In *hydrocephalus interna* the fluid accumulates within the ventricles of the brain, and distends the brain to a great extent. There are two varieties of *hydrocephalus interna*. One is called *congenital hydrocephalus*, and the accumulation of fluid in the ventricles appears to be due to a chronic inflammation of the lining of the ventricles, just as in chronic peritonitis a large amount of fluid may accumulate in the peritoneum. After death, the lining of the ventricles is found to be thickened and rough.

The other variety of *hydrocephalus interna* is called *acquired hydrocephalus*, and it is due to compression of the venæ Galeni, by tumours or by thickening and contraction of the pia mater. The consequence of this compression is a passive exudation of serum into the ventricles, just as ascites results from constriction of the portal vein. This variety is the most common; and if it occur in infancy, the ventricles become enormously distended with fluid, the brain substance thinned out, and the convolutions flattened. The whole cranium becomes enlarged in consequence. In adults no such distension is possible, and death occurs from compression comparatively early.

Symptoms.—The disease usually occurs in infancy, and is characterized by enlargement of the head, with widening of the sutures and fontanelles. If the sutures have recently closed, they may be reopened by the pressure. In extreme cases the head may be enormous, and may even be translucent, if a strong light be placed behind it. The face appears very small, and the frontal bones

project far beyond the eyebrows. But the eyeballs are prominent, because the pressure of the fluid depresses the orbital plates at the base of the skull.

The intellect may be normal, but is more often defective, and the motor power is weak. In bad cases the muscles cannot support the head. Sometimes attacks of vomiting or of convulsions occur, and squinting is common.

A few of these patients live to grow up, and in slight cases the fontanelles may become closed by the formation of ossa triquetra.

Treatment.—Very little can be done for these cases. Cod-liver-oil, phosphates, iron, etc., may be given to improve the general health. Mercury and iodide of potassium may be tried as absorbents. The injection of iodine into the ventricles has been tried, but has not been very successful.

Diagnosis.—At an early stage it may be difficult to distinguish hydrocephalus from rickets, as in both the head is large and the forehead prominent. If the rest of the body shows rachitic changes, if the cranial bones have thickenings on them, if the intellect is good and the eyeballs not prominent, rickets may be safely diagnosed, however gaping the fontanelles may be. In the later stages the great enlargement of the head makes the diagnosis easy.

CONVULSIONS—ECLAMPSIA.

These terms are used synonymously.

Convulsions consist in involuntary clonic (see p. 12) contractions of the muscles, the resulting movements being jerky and purposeless. In some

cases, *e.g.*, epilepsy, tonic contractions of the muscles also occur.

Causes.—1. Convulsions may be caused *reflexly* by irritation of the gums during dentition, or of the intestines by worms. Ovarian and uterine irritation are also reflex causes, and there are rare cases recorded in which slight irritation of the skin and other parts appears to have set up convulsions.

2. The *cerebral* causes of convulsions are of two kinds :

(a) *Lesions*, such as tumours, abscesses, meningitis, etc., of the motor cortex (p. 11) or pons. In these cases the convulsions are often limited to one or two limbs, and the condition is called *Jacksonian epilepsy*.

(b) *Blood conditions*.—Anæmia of the brain, whether due to diminished blood-supply (*e.g.*, obstruction of the arteries) or to impoverished blood (*e.g.*, anæmia or hydræmia), leads to convulsions. Poisons in the blood, such as uræmia and the poisons of the specific fevers, etc., also cause convulsions.

3. *Epilepsy* is a disease characterized by convulsions, but we do not know how they are brought about.

The nervous system of children and of puerperal women is comparatively unstable, and slight causes (such as, in the case of children, teething or the onset of measles) may suffice to cause a convulsion. The chief cause of puerperal convulsions is uræmia, but hydræmia and irritation at the uterus are also causes, the patient being predisposed by the excitability of the nervous system. The tendency to

convulsions is hereditary, and in some families all the children get convulsions during dentition.

Symptoms.—In children the convulsive movements are most common in the eyes, which may be rapidly turned in various directions. The eyelids and muscles of the mouth may also be rapidly contracted and relaxed. In other cases the movements affect the limbs (especially the hands and feet) and in fact the whole body, and may be preceded by a scream, and by a tonic contraction of the muscles, just as in epilepsy. In the graver cases there is complete unconsciousness. The pupils may be contracted or dilated, and sometimes unequal. On coming out of the fit the child is very drowsy.

In Jacksonian epilepsy the convulsive movements are often limited to one arm, or one arm and the corresponding side of the face or leg. But in the case of a large lesion in the pons or medulla, the convulsions may be general. The presence of the symptoms which indicate the nature of the lesion, such as optic neuritis, etc., helps the diagnosis.

Terminations.—Children may die in the fit, but recovery is much more common. Frequently-recurring fits are, of course, dangerous.

If a child four or five years old gets a convulsion, it is very difficult to say whether the case is one of true epilepsy or not; but if there is no family history of epilepsy, insanity or other neurosis, and if the fit does not correspond typically to an epileptic fit, as described below, and if any of the causes of convulsions mentioned above can be discovered, it may be considered, provisionally, as simple eclampsia.

Treatment.—Infantile convulsions may be treated by a hot bath and the application of cold to the

head. If the convulsions are frequently repeated, bromide of potassium (gr. v.) and tincture of belladonna (℥ v.) should be given in aq. chloroform. (ʒi.). In bad cases, chloral hydrate (gr. v.) may be tried, and it may even be necessary to put the child under chloroform. But in all cases the cause should be sought for, particularly in the teeth and the state of the bowels. For instance, lancing the swollen gums or a purgative may stop the attacks. In weak children cod-liver-oil, iron, or the phosphates must be prescribed.

EPILEPSY.

This disease is characterized by the occurrence from time to time of convulsive attacks. No cause can be assigned for the occurrence of the fits at any particular time.

The following are the symptoms of a typical *epileptic fit*: There may be some symptom immediately preceding the muscular contraction. This is called the *epileptic aura*, and it may be a feeling of numbness or of tingling in the fingers, which sensation runs up the arm to the neck, or it may be a disturbance of vision or hearing. Sometimes there is no aura. A flash of pallor across the face can often be observed. The patient may utter a cry, and then falls to the ground, quite *unconscious*, and with all his muscles in a state of *tonic contraction*, so that the limbs may be rigidly flexed, and the head and body twisted to one side. The respiratory muscles are also contracted, so respiration ceases, and the patient becomes deeply cyanosed. The pupils are dilated. This tonic stage lasts only a few seconds, and then the muscles begin to con-

tract and relax rapidly, which constitutes the stage of *clonic spasms*, during which the features work horribly, and the limbs and body may be jerked about. The tongue also moves, and is often bitten severely, and the urine and fæces may be discharged involuntarily. This stage lasts a variable number of seconds, or even minutes, and then the movements cease, the patient slowly regains consciousness, but remains drowsy for some hours.

Epileptics whose attacks are like the above are said to have the *grand mal*. Attacks of lesser intensity constitute the *petit mal*.

In the *petit mal* there may be no convulsive movements, but merely sudden attacks of unconsciousness, with, perhaps, hallucinations of vision or hearing. Sometimes the patient, although unconscious to his surroundings, can yet speak or move, and he may perform acts which may be natural or incongruous, or even dangerous to himself or others. It is maintained that crimes are often performed by persons who are in this state, and really unaccountable for their actions.

If epileptic attacks of either kind are infrequent, occurring, for instance, once or twice a year, the prognosis is not bad. In some cases, even, the tendency to the fits may pass off. But when the fits are frequent, the patient usually becomes apathetic, and incapable of thinking or acting to any useful purpose. Other cases become excitable or vicious.

The *status epilepticus* is a condition in which the patient passes from one epileptic fit into another, so that the spasms are continuous. The patient usually dies in the convulsions. These cases may be distinguished from the continuous convulsions of hysteria by the fact that the temperature rises.

Pathology.—No lesion of any part of the nervous system has been constantly found in epilepsy. Cases in which tumours, sclerosis, etc., have been found can now be placed among the cases of convulsions due to lesions. So epilepsy must be classed, for the present, with the neuroses or functional nervous disorders, and we can only say that epileptics have a peculiarly irritable condition of the nerve-cells (probably those of the medulla), and that at times, from causes which we do not know, some kind of discharge of nerve force occurs from these cells, and produces the seizure.

It is probable, however, that at the onset of the attack there is a transient anæmia of the brain due to spasmodic contraction of the arteries.

Epilepsy is directly hereditary, and also occurs in families in which insanity, dipsomania, and diabetes are prevalent.

Treatment.—The drugs useful in stopping the frequency of the fits are the bromides of potassium and ammonium, paraldehyde (3ss.), oxide of zinc, belladonna, and nitrate of silver. During the fit the patient must be protected from injury by cushions, etc., and a cork or rolled bandage should be put into the mouth to prevent biting of the tongue. Tight clothing should be loosened. Chronic epileptics should be warned against sitting over the fire, as the fit may precipitate them into it.

CHOREA—ST. VITUS' DANCE.

Pathology.—This disease is most common in children from eight to fourteen (especially girls), but may occur in young women, especially if they be pregnant. It is associated with acute rheumatism,

for in very many cases the patients themselves or their parents have had that fever. In most cases a systolic bruit at the apex of the heart may be heard, which, although usually due to endocarditis, may, however, be functional.

No constant lesion has been found in the brain. In all probability the disease is merely due to a weak condition of the motor centres, for children attacked with it are usually weak in body as well as nervous in disposition. It has been suggested that the affection is due to very minute emboli, which come from vegetations on the valves of the heart, and lodge in the motor centres of the brain.

Symptoms.—The disease consists in involuntary movements of the muscles. It occurs in attacks which last six or eight weeks, and may recur two or three times during the course of four or five years. The attacks may be induced by fright or by excitement, even of a pleasurable kind. They usually begin gradually, the child being noticed to grimace, or to fidget, or to drop things. The movements are usually at first most marked on one side of the body (hemichorea), but soon become general. In bad cases the features are constantly working, and the hands are always on the move, the movements suggesting that a voluntary attempt is being made to perform some action, but that the attempt is interfered with by irregular action of the muscles. The movements are on a large scale. If sitting, the legs are kept moving, and if standing, the patient constantly shifts from one leg to the other. In walking, the legs are dragged along the ground. If asked to show the tongue, the patient, after a pause, thrusts it out with a jerk, and quickly retracts it. The movements are worse during ob-

servation, and cease during sleep. The urine has a high specific gravity, and, as already mentioned, there is usually a systolic bruit at the apex of the heart.

In very bad cases the patient cannot sit or stand, and is tossed about the bed by the movements. Death may even occur from exhaustion, and the inability to take food. The mental functions are not impaired in this disease, but in the very bad cases the child looks stupefied.

In a few cases the attack developes to its full extent very suddenly. It always passes off gradually. Death is very rare, and is usually preceded by coma, and sometimes by high pyrexia.

Treatment.—Liq. arsenicalis (℥ iii.) should be given three times a day. At the same time cod-liver-oil, iron, and phosphates should be given to improve the general health. Aperients may be necessary. Fresh air is essential, and all lessons and exciting occupations must be stopped. The patient must be kept away as much as possible from other children, as the latter may develop clonic movements, at first from mimicking, but afterwards involuntarily.

HYSTERIA.

This is a neurosis, *i.e.*, a disease in which various nervous symptoms occur without there being any lesion in the nervous system, or in the parts affected, to account for it. Nearly every symptom of every known disease may occur, at one time or another, in cases of hysteria, and the period during which these symptoms continue, or are liable to occur, may be a few months or many years.

Hysterical patients, either from defects in their strength of will or from self-concentration, exaggerate any pain, or weakness, or emotion; so that for them the pain is excruciating, the weakness a complete paralysis, and the emotion quite uncontrollable. They are eager for sympathy, and, if encouraged, the more severe cases will not only imagine new symptoms, but even create them, as, for instance, by sucking the skin to produce hæmorrhages, etc.

Hysteria is most common in women, and may begin at the age of puberty. It occurs occasionally in men. It is hereditary, and is found sometimes in families in which insanity and other neuroses are prevalent. Many cases set in after some grief, or anxiety, or real illness. Among causes, masturbation, celibacy, and uterine or ovarian diseases have been suggested. Morbid literature is certainly responsible for some cases.

Manifestations of Hysteria.

1. **Motor.**—(a) *Paralysis*, which may affect the lower limbs, producing parâplegia; or the larynx, producing aphonia; or the bladder, producing retention; or the œsophagus, producing inability to swallow; or any muscle or group of muscles.

(b) *Spasm*, which may affect the œsophagus, producing dysphagia, or any muscle or group of muscles. The most serious motor manifestation is *the hysterio-epileptic fit*, in which the patient is suddenly thrown into a convulsion, mostly tonic (with opisthotonos or other contortion), but with clonic spasms at times. The movements differ from those of true epilepsy in being more purposeful; for instance,

the patient may pull out her hair or grip something. During the fit the patient appears to be unconscious, and is at any rate quite anæsthetic. The fits can be produced by deep pressure in the ovarian region. The common hysterical attack of laughing and crying is less serious.

2. **Sensory.**—(a) *Pain* is very common, particularly in the head, or back, or in the knee, or in the ovarian region, where there may be tenderness as well. In the forehead the pain is compared to the boring of a nail (*clavus hystericus*).

(b) *Paræsthesia*.—The *globus hystericus* is a feeling as of a ball in the throat, which can neither be swallowed nor ejected. There may be morbid sensations in the breasts or epigastrium.

(c) *Anæsthesia* is not uncommon, particularly hemianæsthesia, sensation being lost over the whole of one side of the body, including the special senses (sight, hearing, smell, and taste). It may be possible to thrust a needle right through the arm without causing pain.

3. **Mental and Emotional.**—In bad cases moral perversion may be present, the patient being deliberately deceitful. More often there is merely mental depression, the patient brooding over imaginary troubles, or immoderately over real troubles. The hysterical attack of laughing and crying is familiar.

As regards the **Organic Functions**, hysterical vomiting and hysterical suppression of urine are well known. These phenomena may appear most serious, but usually do not affect the patient's health much. The urine is usually pale, abundant, and of low specific gravity. As regards nutrition, in bad cases there may be extreme emaciation.

Treatment.—The antispasmodic drugs, such as valerian, asafoetida, bromides, etc., are not of much use. Any real-disease, if found, must be treated, particularly anæmia by iron, dyspepsia by rhubarb, ammonia, and peppermint-water, and constipation by regular enemata. The hysterical fit can usually be stopped by dashing cold water on the patient or giving her a galvanic shock. But the hysterico-epileptic attack usually cannot be arrested, but runs its course, lasting intermittently, perhaps, for some hours.

Good food and fresh air are, of course, necessary. In bad cases the patient must be sent away from sympathizing relatives and her usual surroundings, and placed among people who will be kind, but firm. Massage gives excellent results in many patients who have become too weak to take proper exercise.

Diagnosis.—No case should be considered hysterical unless every possible organic disease has been carefully excluded by examination. The fundus oculi should be examined for lesions which indicate brain disease, the muscles for any change in the electrical reactions, etc. Patients often use fanciful terms to describe their symptoms, and betray the real nature of the case by the zest with which they enumerate their sufferings.

HYPOCHONDRIASIS.

This is a condition in which the patient, who is usually a man, imagines, without cause, that he has some disease, or attaches grave importance to some trifling symptom. For instance, it may be impossible to convince him that a buzzing in the ears, due to catarrh of the Eustachian tube, is not an indication of an approaching fit or

apoplexy, or that palpitation due to dyspepsia is not caused by serious heart disease. In many cases there is high arterial tension.

MIGRAINE.

This is a form of headache the pathology of which is very obscure. It has been attributed to an excess of uric acid in the blood, which poisons the brain, or, at any rate, produces high arterial tension in that organ. It has also been supposed to be due to a functional affection of the liver. Other suggested causes are anæmia of the brain, or, on the other hand, congestion of the brain. The exciting causes of an attack are: exposure to cold; a vitiated atmosphere, such as is common in theatres; excessive eating, drinking, and smoking; straining of the eyes, such as is necessitated by visiting a picture-gallery; great mental application, especially reading, etc. Some cases supposed to be migraine are due to defective visual accommodation, and can be readily cured by the use of spectacles. Migraine is most common in young adults or in women at the climacteric. Anæmia predisposes to it.

Symptoms.—The name migraine, or hemicrania, is derived from the idea that the pain only affects half the head, *i.e.*, is on one side only. This is often, but not always, the case. The pain is not acute, but rather a dull, unpleasant sensation inside the head, which is greatly aggravated by stooping. It is most often frontal in position, and is accompanied by great depression of spirits; but in many cases the attack is preceded by a feeling of exhilaration, such as sometimes precedes gout. In bad cases there is intolerance of light, and shivering,

with cold extremities and double vision, or even convulsive movements of the facial muscles may occur. Giddiness is common, and vomiting may take place. In the slighter cases the appetite may not be impaired. The attacks may last twenty-four hours, or even for two or three days.

Treatment.—Antipyrin is very useful in these cases. The dose should be small, *e.g.*, 5 grains to begin with, because a small dose often gives complete relief, and 10 grains may produce great depression. Further, the drug may after some time lose its effect, and then it is useful to be able to increase the dose. Phenacetin and citrate of caffein are also of great service. Some cases are relieved by drugs that act on the liver, such as calomel or podophyllin.

CATALEPSY.

This is a rare affection, in which the patient appears to lose consciousness and volition, and the muscles become rigidly fixed in any position in which they may happen to be at the time of the seizure, or in which they may be placed by a bystander. The muscular power would appear to be very great, as the muscles may be able to resist enormous pressure put on them. The condition is mostly hysterical, and is allied to that produced by the hypnotizer or mesmerist.

ATHETOSIS.

This affection is characterized by continuous movements of a peculiar nature. The hands are often affected, and the favourite movement then is a rapid alternate extension with abduction and flexion with adduction of the fingers. Sometimes the

shoulder muscles are affected, and keep the shoulder-joint in constant rotation. Athetosis may occur in a hemiplegic arm, and is then called *post-hemiplegic chorea*. Some cases appear to follow an injury, which has probably damaged the motor nerves.

MÉNIÈRE'S DISEASE.

Some authorities consider that this disease is a neurosis. It is usually, however, supposed to be due to chronic inflammation of the internal ear, or to other lesions, such as hæmorrhages, etc., in that position. Increased pressure on the stapes from the tympanum has also been suggested as a cause.

There are three symptoms: (1) *Deafness*, even to the tuning-fork placed on the forehead. (2) *Giddiness*, which may be so extreme that the patient cannot stand, but reels about, and may tend to fall forwards or backwards or laterally. Even sitting may be impossible, and vomiting often occurs. The giddiness usually comes on in intermittent attacks. (3) *Noises in the ears or head*. These may be like whistling, or the rattling of a train, or like a steam-engine. They may be practically continuous.

Treatment.—Bromide of potassium is recommended.

MENTAL DISEASES.

It will be only possible to refer briefly to these affections. They are all strongly hereditary.

Idiocy and Imbecility.—An *idiot* is a person who has no intelligence whatever, and has never possessed any, so that idiocy dates either from birth or from a period in infancy before any signs of intelligence can be shown.

An *imbecile* is a person whose intelligence is defective. Consequently the degree of imbecility varies very much, the bad cases being little better than idiots, while the slight cases may merely be stupid, and unable to learn anything that is not very simple. Some imbeciles are very mischievous or vicious. In other slighter cases the intelligence, although generally defective, may be highly developed in some particular direction; for instance, they may be able to learn and remember an extraordinary amount of poetry.

Causes.—In most cases of idiocy and imbecility there are structural defects in the brain. These may be *congenital*, resulting from maldevelopment of the brain, or from inflammation, etc., occurring during foetal life, or they may be *acquired*, and be due to cerebral hæmorrhage (occurring during delivery or during an infantile convulsion), or hydrocephalus, or other brain lesions. Repeated epileptic fits eventually lead to imbecility, and in old people the atrophy of the brain produces the same result.

Children who are both blind and deaf are often defective in intelligence from being unable to acquire information.

Bodily Defects.—Most of these cases have deformities, resulting sometimes from the lesions of the nerve-centres. For instance, the head may be small (microcephalic) from undevelopment of the brain, or large from hydrocephalus. The limbs may be rigid from descending sclerosis (p. 21). Other deformities are peculiar shapes of the ears, fingers, and toes, a very highly-vaulted palate, and large everted lips. The sexual organs may be imperfectly developed. These latter defects probably result from the transmission, in an intensified form, of some hereditary tendency to deformity of the ears, fingers, etc., because imbeciles are often the result of consanguineous marriages.

Cretinism.—A cretin is an idiot or imbecile in whom there are certain definite deformities. The disease is usually *endemic*, *i.e.*, it is found in a considerable number of persons who live in a particular locality; for instance, in certain parts of Switzerland. But it may be *sporadic*, *i.e.*, isolated cases may be met with in localities where it has hitherto been unknown.

The points to be remembered about cretinism are: (1) the mental condition, (2) the bony deformities and their consequences, and (3) the condition of the thyroid gland.

(1) The *mental condition* is that of idiocy or imbecility.

(2) The *bony deformities*. (a) Cranial: The ossification of the sphenoid to the occipital bone takes place during intra-uterine life. This prevents the head expanding in an antero-posterior direction,

whence the head of cretins is short antero-posteriorly, and broad transversely. It follows that the forehead is very wide, the eyes wide apart, and the nose flat. Another consequence of this early ossification is that the brain cannot develop to its full size and shape, which explains the mental defects. (b) The Long Bones: These do not develop properly, so that cretins are dwarfs.

(3) The *thyroid gland*. This may be absent, and cause the cretin to be myxœdematous, with thick *alæ nasi*, eyelids and lips, and a thick, harsh, dry skin. In other cases there is a large, but probably degenerated, thyroid.

Sporadic cretinism may develop several years after birth. Fatty deposits in the posterior triangle of the neck have been observed in these cases.

The causation of cretinism is not known. The disease is hereditary, and families afflicted by it die out after four or five generations. Healthy families who come to reside in districts where it is endemic develop the disease at about the third generation.

Insanity.—This consists not so much in a defect as in a perversion of the mental functions. The great characteristics of insanity are: (1) a change in the disposition of the patient, the cheerful man becoming morose, the tidy man slovenly, etc.; and (2) the presence of delusions, the patient imagining himself to be someone else, or complaining of imaginary wrongs, or hearing imaginary voices, etc. Insanity is strongly hereditary, not only to itself, but to alcoholism, epilepsy, and other neuroses.

The following are the chief forms in which insanity may occur:

1. **Acute Mania.**—In this condition the patient

raves incoherently, making use of pious or foul expressions in a loud tone, and with rapid articulation. He is usually violent in his movements and gestures, and may threaten to injure himself or others, and often succeeds in doing so. He will neither eat nor sleep for long periods. The attacks may be at long intervals or frequent. In the latter case death usually takes place.

2. **Melancholia.**—In this condition the patient is apathetic, and may sit for hours, noticing nothing and not replying to questions. If he speaks, it is in a mournful tone, and merely to repeat the story of his woes. He may imagine himself about to die, or predestined to damnation, or persecuted by imaginary enemies. He often refuses food. These cases are of longer duration than those of mania.

3. **Monomania.**—In this condition the patient is mad on one subject, though the term is not used with absolute strictness. The uncontrollable desire to kill (homicidal or suicidal mania), to steal (kleptomania), etc., are instances of this form.

4. **General Paralysis of the Insane.**—The two points to be considered here are (*a*) the mental state and (*b*) the paralysis.

(*a*) The mental state is characterized by exaltation of ideas, or *megalomania*. The patient imagines that he is a deity, a millionaire, a genius, etc., and he will endeavour to act on these ideas. For instance, he may order goods to the value of hundreds of pounds, while his income is really a hundred a year. Sometimes mania or melancholia may be present.

(*b*) The muscles become weak and tremulous, the condition being paresis rather than paralysis. The first muscles affected are usually those of the

lips and tongue, so that a quivering of the lips on attempting to speak, and a hesitation or thickness in articulation, are early signs. The disease is progressive, and month by month other muscles are affected, becoming wasted and weak. Eventually the patient becomes too weak to move, and death occurs in three or four years.

The muscular symptoms resemble those of bulbar paralysis (see p. 66) and of disseminated sclerosis, and in many cases sclerosis is found in the medulla and scattered through the brain and spinal cord. In particular, sclerotic patches may be found in the gray cortical matter of the brain. In some cases, more or less organized fibrinous layers are found on the dura mater, with perhaps hæmorrhages between the layers, a condition which is called pachymeningitis.

Treatment.—Careful feeding and the administration of sedatives during excitement constitute the chief treatment.

DISEASES OF THE SPINAL CORD.

LOCALIZATION.

THE functions of the following parts of the spinal cord must be remembered :

1. *The multipolar cells in the anterior cornua* preside over the motor power, the nutrition, and the electrical reactions of the muscles. When they are destroyed, the muscles become paralyzed and wasted, and give the *reaction of degeneration* (see p. 14).

The diseases in which these cells are specially affected are infantile and adult spinal paralysis and progressive muscular atrophy. The words antero-polio-myelitis mean inflammation of the anterior cornua (gray matter) of the spinal cord.

In the *medulla oblongata* the nuclei of origin of the cranial nerves are formed by the same kind of multipolar cells as those of the anterior cornua of the cord, so that when they are completely destroyed by lesions such as sclerosis (bulbar paralysis) or tumours, the muscles presided over by them become paralyzed and wasted, and give the reaction of degeneration.

2. *The crossed pyramidal tracts in the lateral column* merely conduct motor impulses which come from the brain. When there is a lesion confined to these tracts, paralysis results ; but if the lesion be irritative (*e.g.*, sclerosis), the paralyzed muscles become rigid.

These tracts are affected in descending sclerosis and spastic paraplegia.

3. The *postero-external columns* appear to preside over the co-ordination of muscular movements. They are the seat of the lesion in locomotor ataxy.

4. The *postero-median columns* (band of Goll) conduct ordinary tactile sensations to the brain. When they are destroyed anæsthesia results. They become the seat of sclerosis when the cord beneath them is destroyed (see below), and they may also be affected in locomotor ataxy.

5. The part of the *reflex loop* (see p. 14) within the cord consists of the posterior and anterior cornua, including the multipolar cells.

The above points may be illustrated by describing the symptoms of a lesion which completely destroys the cord for an inch or so of its length. Such destruction may be due to the pressure of a dislocated vertebra, or of a tumour, etc. As soon as the cord is destroyed, all the parts whose sensory nerves reach the cord at or below the site of the lesion become *anæsthetic*, because the sensory impressions originating in them cannot reach the brain. Also all the muscles whose nerves come off at or below the site of the lesion become *paralyzed*, because no impulses from the brain can reach them. This constitutes *paraplegia*. Now the muscles whose nerves come off from the site of the lesion become *flaccid*, rapidly *waste*, and give the *reaction of degeneration*, because their multipolar cells are destroyed; and any *reflexes* in which they are concerned are *lost*, because their reflex loop in the cord is destroyed. But the muscles whose nerves come off below the lesion do *not waste nor lose their electrical reaction*, because their multipolar cells are intact; and any

reflexes in which they are concerned are *exaggerated*, because, while their reflex loop is intact, they are cut off from the inhibitory action of the brain by the destruction of the cord above. Further, it usually happens that sclerosis affects the crossed pyramidal tracts below the lesion, and then these muscles become *rigid*. Hence, by observing the electrical reactions and the state of nutrition of the paralyzed muscles, and the condition of the reflexes, the position of the lesion in the cord can be ascertained, and if it be removable by operation (*e.g.*, a dislocated vertebra), it can be cut down on. Lastly, the *bladder-centre* is important. If it be destroyed, the bladder does not contract at all, the urine is retained or dribbles away when the bladder is overfull, and, if a catheter be passed, much of the urine must be pressed out. But if the bladder-centre be normal, but cut off from the brain by the lesion, then the bladder acts involuntarily, and if a catheter be passed, it contracts well on its contents. The sensory fibres in the postero-median column above the lesion undergo sclerosis.

LESIONS OF THE SPINAL CORD.

The cord may be affected by inflammation, hæmorrhages, tumours, etc., and it is very liable to compression by diseased or dislocated vertebræ. The nature of the lesion is more difficult to diagnose in the cord than in the brain. We can only say that very rapidly occurring symptoms would indicate a hæmorrhage, that acute inflammations cause symptoms in a few hours, that tumours produce symptoms in a few weeks, and chronic inflammation (sclerosis) in the course of weeks or months.

ACUTE ANTERO - POLIO - MYELITIS. — INFANTILE PARALYSIS.

The cause of this disease is unknown. It is most common at about two years of age. The lesion is an acute inflammation of the multipolar cells in the anterior cornua, which ends in the complete destruction of these cells, and a total abolition of their functions. There is, probably, also a little hæmorrhage into the anterior cornua. When death occurs years after the occurrence of the disease, the affected part of the anterior cornu is found to be shrunken and sclerosed (see below), with perhaps some pigment in the sclerotic tissue, while the multipolar cells are absent or degenerated.

Symptoms.—The disease begins with slight fever, which may be attributed to a cold, or to the approach of measles, etc. There may be a convulsion. Then, in the course of a few hours, there is paralysis of a group of muscles—sometimes the whole of the leg or arm, or sometimes only a few muscles in either limb, or even in the trunk. In a few days the paralyzed muscles begin to waste, lose their faradic contractility, and give the reaction of degeneration, A. C. C. becoming greater than C. C. C. (see p. 14). It is usual, however, for some of the muscles to recover, probably because the multipolar cells connected with them have been merely damaged, but not destroyed by the lesion, and the muscles which are recovering can be distinguished from those that are hopelessly paralyzed by the fact that the former do not give the reaction of degeneration. Sensation is never lost.

As time goes on, the paralyzed muscles waste until they become mere fibrous bands, with or without some fatty deposit. This may lead to

deformity; for instance, if the muscles on the front of the leg are affected, the unopposed action of the calf muscles causes talipes equinus. If a large group of muscles be affected, *e.g.*, the whole leg, the skin of the leg is cyanosed and cold, and the bones do not grow, so that the limb is short and small. The knee-jerk would, of course, be lost.

This disease occasionally occurs in adults, and is called *adult spinal paralysis*. No treatment is of any use, but massage and galvanization of the affected muscles should be tried.

SCLEROSIS.

Causes. — Syphilis, alcoholism, gout and the uric acid diathesis, sexual excess, and concussion. Sclerosis also occurs as a secondary change. For instance, if the cord be completely crushed at one point by a dislocated vertebra, the motor tract in the lateral columns below the crushed part can no longer carry motor impulses from the brain, and it then becomes sclerosed. Also the sensory tract in the postero-median columns above the crushed part can no longer carry sensory impressions to the brain, and it also becomes sclerosed.

Pathological Anatomy.—There are two processes at work in sclerosis. One is the degeneration of the nerve cells and fibres. The *cells* slowly lose their nuclei and their processes, and become irregular masses or disappear. In the *fibres* the nuclei of the primitive sheath become enlarged, and the myelin breaks up into droplets and is absorbed. The axis cylinders persist for a long time.

The other process in sclerosis is the overgrowth of the neuroglia. In the gray matter the increased neuroglia replaces the degenerated cells, and in the

white matter it separates the nerve-fibres and constricts them. In all probability, the degeneration of the nerve tissues is the primary change.

Sclerotic nerve tissue is gray, hard, and contracted like a scar. It is stained deeply by carmine, because of its increased connective tissue, and faintly by osmic acid, because of its diminished myelin. Microscopically, in the white matter, the nerve-fibres are seen to be few and unequal in size, and separated by the increased neuroglia. Droplets of myelin may also be seen and large corpuscles, derived from the nuclei of the primitive sheath.

Sclerosis in the medulla causes *bulbar paralysis*; in the anterior cornua, *progressive muscular atrophy*; in the lateral columns, *late rigidity* and *spastic paraplegia*; and in the postero-external columns, *locomotor ataxy*. In *disseminated sclerosis* it occurs in small patches all over the brain and cord, and it is commonly found in the cerebral cortex in *general paresis of the insane*.

Treatment.—It is usual to give potassium iodide and mercury in the hope that the sclerosis is syphilitic. Strychnia hypodermically and tonics of all kinds may be tried. Counter-irritation, even by the actual cautery, has been recommended.

Bulbar Paralysis—Glosso-Labio-Pharyngeal Paralysis.

Lesion.—Sclerosis of the nuclei of origin of the facial, hypoglossal, glosso-pharyngeal, and vagus nerves in the medulla.

Symptoms.—The disease begins with wasting and weakness of the muscles of the lips and tongue. This interferes with speech, particularly with the

pronunciation of the letters *o* and *u*, and *p* and *b* by the lips, and *th*, *r*, and *s* by the tongue. The lips often quiver when an attempt is made to speak. The individual fibres of the muscles waste, almost one by one, so that the reaction to faradization is slowly less and less, while that to galvanization gradually gets more marked, and A. C. C. gradually gets greater than C. C. C. (see p. 14).

As the tongue gets worse the soft palate gets affected, so that the voice becomes nasal, and food returns through the nose. The lifting of the palate can be seen to be weak. The buccinator also atrophies, causing the food to collect between the teeth and the cheek.

Then the pharynx is taken, and swallowing becomes difficult. In particular, food may get into and obstruct the larynx, causing immediate suffocation. The laryngeal muscles may also become weak, causing aphonia. By this time the atrophy of the muscles which were first affected is extreme. The lips are thin, motionless, and do not meet; the cheeks are smooth, and the tongue a shrivelled, motionless mass at the floor of the mouth.

The levator anguli oris is affected late, so that the patient may be able to grin. The orbicularis palpebrarum may not be affected at all, the reason being that, whereas the fibres of the seventh nerve which go to the mouth come from a nucleus which is close to that of the hypoglossal, the fibres which go to the eye come from a nucleus which is close to that of the third nerve in the pons, and therefore is a long way from the place where the lesion usually begins.

Bulbar paralysis may coexist with the other diseases produced by sclerosis, particularly with

general paresis. The medulla is also the seat of many patches in disseminated sclerosis. Death usually occurs in two or three years.

Progressive Muscular Atrophy—Chronic Antero-Polio-Myelitis.

Lesion.—Sclerosis of the multipolar cells in the anterior cornua of the cord. It usually begins in the cells which are connected with the nerves of the arm.

Symptoms.—The muscles of the thenar and hypothenar eminences of the right hand become wasted and weak. Then the interossei are affected, and then the corresponding muscles of the left hand. There are often fibrillar twitchings in the wasting muscles. The paresis and the diminution in the reaction to faradization are proportional to the wasting; but as the individual fibres of the muscles atrophy one by one or in groups, a contraction to faradization can be obtained from those which remain for a long time. At a certain period the reaction to galvanization increases, and A. C. C. becomes greater than C. C. C. Sensation is always normal.

The disease is progressive, the muscles of the forearm being next taken, the supinator longus going early. Then the upper arm atrophies, the triceps often escaping for a long time. The deltoid is usually affected early, but the anterior part of the trapezius very late.

The progress of the disease goes on through many years, and it may even appear to be arrested for a year or two. In old cases the hand becomes like a claw, being hollowed out by the atrophy of the

palmar muscles, and rigid from fibrous contraction. Death eventually results from bronchitis, after the respiratory muscles have become weak. Occasionally the disease begins in the shoulder.

Diagnosis.—This disease is distinguished from *lead palsy* by the facts that in the latter there is pain, the extensors are more affected than the flexors, and the supinator longus is affected late. As regards *infantile paralysis*, it will be remembered that in that disease the muscles become paralyzed at once, and then waste, while in progressive muscular atrophy the weakness and wasting proceed together, because the sclerosis creeps from one multipolar cell to another, causing the atrophy of separate fibres of the muscles as each cell is destroyed.

Microscopically, in both bulbar paralysis and progressive muscular atrophy the muscular fibres have lost their striation, and are filled with granules.

Treatment.—In addition to that for sclerosis, the affected muscles in both diseases should be shampooed and galvanized.

Lateral Sclerosis.

Varieties.—Sclerosis in the crossed pyramidal tracts of the lateral columns may be secondary or primary. If *secondary*, it may follow a lesion in the motor tract of the brain, in which case it affects one side of the cord, and causes *late rigidity* (see p. 20), or it may follow a spinal lesion (such as the destruction of the cord by a dislocated vertebra, or a tumour, see p. 65), in which case it affects both sides of the cord, and causes *spastic*

paraplegia. Both secondary forms are called *descending sclerosis*, because the sclerosis appears to descend from the primary lesion along the motor paths.

If *primary*, lateral sclerosis may be due to syphilis, etc., and may affect one crossed pyramidal tract before the other. When it affects both sides, a spastic paraplegia results.

Symptoms—Primary Lateral Sclerosis.—The effect of the sclerosis in the motor path is to make the muscles weak and rigid, and to exaggerate their reflexes. The legs are first affected. The flexor and adductor muscles being the most powerful, when they become rigid the former cause the toe to be rigidly pointed, and the latter draw the legs to the middle line. Hence walking is difficult, the toe being often *stumped* against small objects on the ground, while the difficulty in separating the legs causes tripping of one leg over the other. Finally, the *gait* is as follows: The hip is raised, so as to get the pointed toe off the ground, for the leg cannot be swung round, as in hemiplegia, because of the rigidity of the whole limb; then one foot is laboriously worked past the other (often touching it on the inner side the whole time, on account of the rigid adduction). When it has got past, the rigidity permits only a short forward step. Progression is, of course, very slow, and the narrowing of the base of support by the adduction causes the patient to resort to the aid of a pair of sticks at an early period. While in action the muscles are often tremulous. The other symptoms of this affection are ankle clonus and increased knee-jerk; and the negative points are that the muscles do not waste nor lose their normal electrical reactions;

that the bladder is not affected, and that sensation is normal.

The symptoms of a tumour slowly growing in the lateral column would be the same as those of sclerosis in that position.

Lateral sclerosis is often associated with other forms of sclerosis. The term *amyotrophic lateral sclerosis* means lateral sclerosis with atrophy of the muscles, and the condition is due to sclerosis of the anterior cornua, which causes the atrophy, plus sclerosis of the lateral columns, which makes the muscles rigid.

Lateral Sclerosis secondary to a Spinal Lesion.—In this case the primary lesion, *i.e.*, the destruction of a transverse piece of the cord, causes immediate paralysis of all the muscles below the lesion (paraplegia), so that the patient cannot walk at all. The plantar reflex is at once increased, and there is anæsthesia below the lesion. When the sclerosis develops, the paralyzed muscles become rigid, but do not waste or lose their electrical reactions, and the deep reflexes are increased; but the condition differs from the primary form in that there is anæsthesia, and that the bladder is affected. Bed-sores often form on the anæsthetic parts. The position of the primary lesion can be ascertained by examining the muscles as described on p. 62.

Locomotor Ataxy—Tabes Dorsalis.

Lesion.—There is sclerosis in the postero-external columns of the cord. It is often syphilitic, and is most common in men. In the upper part of the cord the postero-median column may also be sclerosed.

Symptoms—Motor Symptoms.—The muscles are not paralyzed, but there is a loss of co-ordination, which is called *ataxia*. This means that the muscular contractions are not combined harmoniously to produce the movements which are desired, so that these movements are irregular. The *legs* are usually first affected. The patient *cannot stand with his eyes shut*, and the *gait* is as follows: The legs are lifted too high, thrown too far outward and too far forward, and then brought down too forcibly on the heel. The difficulty in walking is first observed in the dark, or on first starting to walk, or on turning quickly. When the *arms* are affected, the patient cannot button his clothes or pick up a pin.

Sensory symptoms are often early. Numbness of the feet is common, the patient feeling as if he was treading on wool. Sensation is really *delayed*, *i.e.*, the brain does not appreciate a sensation until a few seconds after it has stimulated the skin. Pains called *lightning pains* often flash along the nerves, and sometimes there is a sense of constriction, called the *girdle pain*, round the waist.

The *muscular sense* is often lost, the patient being unable to tell the position of his limbs or to distinguish weights when his eyes are bandaged.

The *reflexes*. The *knee-jerk*, and the *plantar* reflex are lost early. The pupils do not contract to light (a reflex act), while they still contract on accommodation. This is called the *Argyll-Robertson* phenomenon.

Eye Symptoms.—The *pupils* are usually contracted, and *ptosis* and *double vision* may be present. *Atrophy of the optic disc* may occur, and cause complete blindness.

Trophic Symptoms.—*Herpes* may occur on the skin supplied by the nerves affected by the lightning pains, and *perforating ulcer of the foot* may be present. A rapid and painless effusion into the knee may occur, and end in disorganization of the articular ends of the bones, so that the joint becomes loose and the leg swings like a flail. This is called *Charcot's knee*, and the shoulder, elbow, etc., may be affected in the same way.

Visceral Symptoms.—Attacks of gastric pain with vomiting, called *gastric crises*, may occur, and there may be a difficulty in starting or in restraining the act of micturition. Sexual power is said to be increased at first, but it is soon lost.

Treatment.—Iodide of potash and strychnia should be given, and nerve-stretching and suspension of the patient by the arms have been recommended. The disease lasts for years.

Friedrich's Disease is a form of tabes which occurs in families, several members of which become affected at about the age of puberty. The legs become ataxic, and the knee-jerks are lost. Nystagmus and stammering speech have also been observed.

Disseminated Sclerosis—Insular Sclerosis.

Lesion.—There are small patches of sclerosis scattered through the brain and spinal cord.

Symptoms.—There are three cardinal symptoms: (1) Tremors of the muscles when they are used to perform a voluntary act—when not in use they do not shake at all. The hands are usually affected first, and the muscles are distinctly paretic. (2) Nystagmus, *i.e.*, an involuntary oscillation of the

eyeballs. (3) Scanning or staccato speech, in which the patient jerks out each syllable, detaching it by a short pause from the next.

In addition, if sclerotic patches occur in the *medulla* (as is common), there are symptoms of *bulbar paralysis*, such as paresis and atrophy of the lips and tongue, etc. (see p. 66). If they occur in the *lateral columns* (as is common), symptoms of *lateral sclerosis*, such as rigidity of muscles, a spastic gait, and increased reflexes, etc. (see p. 70), supervene. If they occur on the *cerebral cortex*, symptoms of *general paresis of the insane*, such as *megalomania*, etc. (see p. 59), occur. As a matter of fact, disseminated sclerosis, bulbar paralysis, and general paresis of the insane, very often occur together. Patches may also occur in the anterior cornua, causing muscular atrophy; in the postero-median column, causing loss of sensation; and in the postero-external column, causing ataxic symptoms.

The disease is usually fatal in a few years, particularly if the sclerosis affect the medulla.

PARALYSIS AGITANS.

This disease resembles disseminated sclerosis in the fact that there are tremors of the muscles, but the two are easily distinguished one from the other, because in disseminated sclerosis the tremors only occur when a voluntary movement is made, while in paralysis agitans the tremors are continuous, occurring during rest. In the early stages the tremors may even be controlled by an attempt at voluntary movement.

Pathology.—No characteristic lesion has been found in the brain, cord, or nerves. The proliferation of the cells in the central canal of the cord which is usually found, is also found in other diseases. There is no sclerosis. The disease is most common in middle-aged women.

Symptoms.—The hands are usually affected first. They become subject to rhythmical tremulous movements, which gradually become continuous. The fingers often move as if they were rolling bread pills. The tremors then extend to other parts of the body. The facial muscles are not affected, but the head is, in many cases, continually in movement, either nodding or moving from side to side.

The other symptoms are rigidity and weakness of the muscles. The head and shoulders are often bent forward. The gait is peculiar, the patient hurrying forward with short steps, as if he were afraid of falling. This is called *propulsion* or *festination*. If while thus hurrying he be pulled back, he will often hurry backwards in the same manner. This is called *retropulsion*. The patient often complains of internal heat, though the temperature is not raised. Sensation is normal, and the mind is unaffected. The disease may last for years, and no special treatment can be recommended.

PSEUDO-HYPERTROPHIC PARALYSIS (DUCHENNE).

Pathology.—There is a slow atrophy of the muscular fibres, which, however, retain their striation until they have almost disappeared. The connective tissue and fat between the muscular fibres

are in most cases enormously increased, so that the muscles appear large, although they are weak from the atrophy of their fibres. There is no lesion in the brain, cord, or nerves.

The disease is most common in male children. It is hereditary, being transmitted by the mother (who is not herself affected, but who comes from an affected stock) to her sons.

Symptoms.—The muscles of the gluteal region and the other extensors of the trunk on the thigh become enlarged, but very weak. This weakness causes a difficulty in walking upstairs, and it leads to very great *lordosis* of the lumbar vertebræ, the shoulders being thrown back to maintain the equilibrium. On sitting down, the lordosis disappears, showing that the back muscles are not at fault. The calves of the legs also become large and weak, so that the patient cannot point the toes, and, as the base of support is weak, he stands with the legs wide apart. The *gait* is waddling on account of the separation of the legs and the lordosis and the swaying of the shoulders.

In getting up from the dorsal decubitus, the patient first turns to one side, places his hands on the ground, and uses his arms to help him to a sitting posture. Then he gets on his hands and knees, and then (by extending the knees) on his hands and feet, so that the gluteal region is the highest point. As he cannot lift up the trunk, he now places his hands on his shins, and, by working them up the legs and thighs, gradually pushes up his shoulders, until he can balance himself in the erect posture. Or he may use a chair or table to help him to lift the trunk.

The electrical reactions of the muscles are normal,

but diminished in intensity. The reflexes also are diminished. The disease extends later to the fore-arms and shoulders, and eventually to the trunk, death occurring when the respiratory muscles become affected.

In some cases no fat develops between the muscular fibres, so that there is no pseudo-hypertrophy. Rarely the disease occurs in adults, in which case the pseudo-hypertrophy is less marked, and the distribution is not the same as in the infantile form, the facial or scapulo-humoral muscles being often first affected. No special treatment can be recommended.

ACUTE MYELITIS—INFLAMMATION OF THE SPINAL CORD.

Pathology.—No cause is known. The disease consists in a rapid softening of the spinal cord. There may be some hyperæmia, but usually the cord appears as a diffuent white substance within its membranes. Microscopically, the multipolar cells are disintegrated, while the myelin of the white substance is broken up into drops. Numerous leucocytes may be seen. In some cases hæmorrhage occurs in the softened tissue.

Symptoms.—(a) If the softening begin at the very bottom of the cord, the first symptoms are *pain* in the feet, *spasm* of the plantar muscles, and *increase of the plantar reflex*, all of which result from irritation of the cord. In from twelve to twenty-four hours there is *anæsthesia* and *paralysis* of the feet, with *loss of the plantar reflex*, resulting from destruction of the cord. By this time there is pain and spasm of muscles in the leg, followed

in due course by anæsthesia and paralysis. In two or three days or earlier the thigh becomes affected; and by this time the muscles of the feet have lost their faradic contractility, and are beginning to waste. In this way, as the softening spreads up the cord, successive parts have their functions transiently irritated and soon lost, and when the respiratory muscles are paralyzed, death occurs, the diaphragm being the last to go. The bladder and rectum are, of course, affected early, and priapism may occur while the generative centre is being irritated. Sometimes the acute bedsore (see p. 16) developes on the sacrum. The application of a hot sponge to the back is said to cause a deep-seated pain.

(*b*) If the softening does not begin at the bottom of the cord, the cord below it may remain normal, while the lesion runs upwards. Then the parts supplied by the nerves which come off below the lesion, although anæsthetic and paralyzed, retain their reflexes (the reflex loop being intact), and the muscles do not waste nor give the reaction of degeneration. If the patient lived long enough, the muscles would probably become rigid from the development of lateral sclerosis below the lesion.

The disease is usually fatal in a few days. No treatment is of much use. The actual cautery may be applied over the spine. Cases, apparently, of this disease have become arrested, but have, of course, left permanent paraplegia.

ASCENDING SPINAL PARALYSIS (LANDRY).

This disease resembles acute myelitis in the fact that there is a rapidly occurring paralysis, first of the feet, then of the legs and thighs, etc., until the

respiratory muscles are reached. The reflexes also are lost. It differs from acute myelitis in the facts that there is no anæsthesia and no affection of the bladder or rectum.

The progress of the disease is sometimes arrested, even after the breathing has become diaphragmatic, and then the muscles slowly regain their power. No constant lesion is found after death.

PARAPLEGIA.

Any condition in which both lower limbs are paralyzed may be called paraplegia, and it will have been noticed that such paralysis may be due to various causes.

For instance, when the cord is crushed by dislocation of the vertebræ by accident or after caries, or when it is destroyed by the pressure of a tumour, or softened by myelitis, or torn up by hæmorrhage; in all such cases there is paraplegia, with loss of sensation and a tendency to bedsores. The muscles may be flaccid or rigid, wasted or not, and may give the reaction of degeneration or not, according as their nerves come off from the destroyed part or from the parts below. The condition of the reflexes and of the bladder and rectum depends on the same circumstance, as explained on p. 62.

In these cases a water-bed should be used, as a precaution against bedsores, and the gluteal region should be scrupulously cleaned, washed with spirit and water, and powdered with zinc oxide. Cystitis with ammoniacal ropy urine is likely to occur, probably because septic organisms enter the bladder in non-sterilized catheters.

Landry's paralysis is also a paraplegia, but there is no loss of sensation.

Sclerosis of the lateral columns also causes paraplegia, with rigidity of the muscles and increase of the reflexes, but without wasting or change in the electrical reactions. There is no loss of sensation, or affection of the bladder or rectum.

Again, neuritis of the nerves of both lower limbs causes paraplegia. This sometimes occurs in chronic alcoholism (see p. 94).

Lastly, hysteria may simulate paraplegia, the patient being unable to use the lower limbs, and sometimes even having paralysis of the bladder.

SPINAL MENINGITIS.

Causes.—It has been attributed to injury, cold, septicæmia, scarlet fever, etc.

Pathological Anatomy.—The surface of the arachnoid on the cord and on the dura mater is covered with fibrin and leucocytes, and the arachnoid cavity contains much serum. Sometimes the fluid is purulent. Beneath the fibrin the pia mater is congested.

Symptoms.—These depend on the fact that the spinal nerves pierce the inflamed meninges on their way out of the vertebral canal, and that they are consequently first irritated and then destroyed.

The irritation of the *sensory roots* causes severe pains in the course of the nerves, either down the legs or arms or round the trunk. There may also be a burning pain in various parts of the skin supplied by these nerves. Towards the end the parts which had been painful become anæsthetic.

The irritation of the *motor roots* causes at first painful contractions of the muscles, which are either transient or permanent. In particular, the back of the neck is usually retracted by muscular

spasm, or even the whole back may be arched (opisthotonos). Later on paralysis may affect these muscles.

The reflexes are increased at first, and afterwards lost. There is always fever of an irregular type, and in addition to the pains along the nerves, there is pain in the back, made worse by movement or by percussion.

Differential Diagnosis.—In acute myelitis the period of pain and muscular spasm is much more quickly followed by that of anæsthesia and paralysis, and the pain in the back may be only brought out by applying heat, and is not made worse by movement. For the diagnosis from tetanus, see p. 83.

Treatment.—Counter-irritation along the back may be used, and mercury and iodide of potassium may be given internally. Opium may be given to allay the pain.

TETANUS.

Pathology.—Although it is convenient to place it among the nervous diseases, tetanus is really a specific disease, due to a bacillus.

The bacillus is often bulbous at each end, from the presence of a spore in that position. It is found outside the body in mould, and it grows readily in all media, producing a poison (toxin), to which the symptoms are due. From cultivations of the bacillus, an antidote to the toxin, which is called the *antitoxin*, has been prepared, and this, when injected hypodermically, is said to cure the disease.

The bacillus enters the body through some wound. It remains in the wound, where it produces

the toxin, which is absorbed into the blood and irritates the spinal cord. The bacilli do not themselves enter the blood. If no wound can be found, the disease is said to be *idiopathic*, but probably in all cases there has been a wound. No lesion of the cord is found after death, but inflammation of the nerves involved in the wound may occur.

Tetanus is more common in tropical climates. It may affect infants (*trismus neonatorum*), probably from infection of the umbilical cord.

Symptoms.—There is, first of all, a painful stiffness of the lower jaw from spasmodic contraction of the masseters and temporal muscles. This gradually gets worse, until in the course of two or three days, or earlier, the jaw may be rigidly and continuously clenched, so that the patient cannot be fed by the mouth. This early symptom has given the name of lockjaw, or *trismus*, to the disease.

At a variable period in the first few days of the disease the tetanic spasms begin to affect other parts of the body. The muscles of the legs and arms may at any moment be thrown into a state of tonic spasm, which may last a variable time. Most often tetanic spasms affect nearly the whole body, so that the patient is thrown into extraordinary attitudes. The back may be so arched (*opisthotonos*) that the body rests on the occiput and the heels, or there may be a bending forward (*emprosthotonos*), or a twisting to one side (*pleurosthotonos*). The contraction of the facial muscles may produce a grin, called the *risus sardonicus*. The contractions of the muscles cause intense pain, like that of cramp, and the muscles have even been known to rupture. The pupils are usually extremely contracted.

The general tetanic spasms usually relax after a few seconds, for otherwise death would occur from spasm of the respiratory muscles. They, however, recur with varying frequency, perhaps two or three a day or an hour. They may be excited by touching the patient, or even by moving about the room. The more localized spasms, such as those of the limbs and back, may be more persistent, and the lockjaw is usually continuous.

The mind is clear throughout the disease, and there may be no fever or other symptoms.

Death may occur at any time in a general spasm. If the spasms are sufficiently infrequent to enable the patient to sleep and to be fed, recovery may take place. Every day the patient survives after the fourth or fifth day makes the prognosis more favourable.

Treatment.—The antitoxin should always be tried as early as possible. Sleep should be procured by giving choral hydrate and bromides; and feeding through the nose by a tube and rectal nutrient enemata may be necessary, the administration of nourishment being an essential point. To subdue the spasms, conium, belladonna, etc., and the production of anæsthesia by chloroform, are recommended.

Differential Diagnosis.—Tetanus must be distinguished from strychnia-poisoning, as tonic spasms occur in both. But in **strychnia-poisoning** (1) the spasms set in suddenly, so that the first one may affect the whole body; (2) the jaw is not affected specially; (3) in the intervals of the spasms there is complete muscular relaxation; (4) if death does not occur, the symptoms pass off in twenty-four hours. On the other hand, in **tetanus** the jaw is

affected for some time before the general spasms begin, and, although the general spasms relax, the spasm of the jaw is continuous.

The rigidity of the muscles of the back may cause spinal meningitis to simulate tetanus. But in tetanus there is only pain when the muscles are contracted and the trismus is characteristic, whereas in spinal meningitis the pain in the back is constant, and the pains shooting down the nerves are characteristic.

HÆMORRHAGES INTO THE CORD.

Hæmorrhages may occur without any obvious cause, but most often the effusion of blood takes place into a piece of cord which has been previously softened by acute myelitis or by gliomatous infiltration.

The **Symptoms** depend on the part of the cord affected. For instance, if the hæmorrhage is in the anterior cornua, paralysis of a group of muscles, with loss of reflexes and of the reaction to faradism, will occur, as in acute antero-polio-myelitis; but these symptoms would develop instantaneously, and not in the course of a few hours, as is the case in myelitis.

Again, if the hæmorrhage destroy the whole transverse thickness of the cord, complete paraplegia will follow, as in acute myelitis, but all the symptoms would develop instantaneously, instead of ascending gradually (see p. 77).

TUMOURS OF THE CORD.

Tubercular tumours, gummata, and sarcomata may grow in the cord. The same tumours and lymphadenomata also may grow from the meninges or bones, and compress the cord.

The **Symptoms** will depend on the position of the tumour. For instance, if it grew in the lateral column, the spastic gait and other symptoms of primary lateral sclerosis (see p. 70) would occur.

In **Syringo-myelia**, a soft pinkish-gray swollen tissue grows around the central canal of the cord. It has the same microscopic characters as a glioma, and may contain ganglionic cells. The central canal may appear dilated, from liquefaction of the growth. On account of its position in the cord, it produces loss of *painful sensations*, which may be so marked that a patient sitting before a fire may burn his legs severely without being aware of it. If the growth spreads laterally in the cord, other symptoms occur.

SPINA BIFIDA—HYDRORACHIS.

This is a congenital disease, in which the spines and laminae of certain vertebræ do not develope, and in consequence the vertebral canal remains unclosed. Nearly always it is the last two or three lumbar vertebræ which are thus incompletely formed. On account of the absence of bony support, the arachnoid cavity becomes distended by cerebro-spinal fluid, and bulges through the opening in the bones, forming a cyst-like tumour in the back. The tumour may be covered by skin, but sometimes the skin is absent, and the covering consists only of the dura mater and the vertebral layer of the arachnoid. The nerves of the cauda equina may be adherent to the sac-wall.

The **Symptoms** consist in the presence of the tumour, but there may be paraplegia. Death usually occurs early, the cyst being very liable to rupture. If it be very tense, paracentesis may be performed.

DISEASES OF THE NERVES.

THE principal affections of nerves are as follows :

1. **Acute Neuritis.**—Causes : Cold ; the toxins of the fevers, especially diphtheria, but also sometimes malaria, typhoid, influenza, etc. ; the involvement of the nerve in a suppurating or gangrenous tissue, *e.g.*, the seventh nerve in necrosis of the temporal bone. Acute neuritis is usually parenchymatous.

2. **Chronic Neuritis.**—Causes : Cold ; rheumatism ; malaria ; alcohol ; lead. Chronic neuritis is usually interstitial—that is, the increase of the neuroglia is the most conspicuous change.

3. **Pressure** by tumours or by periosteal or bony thickenings. The last occurs at foramina, through which the nerves pass ; *e.g.*, branches of the fifth nerve may be compressed by such thickenings in the foramina through which they pass to the face.

4. **Tumours** in the course of the nerves. Sarcomata, gummata, and tubercles may grow in a nerve, and fibromata and myxomata also occur.

5. **Injury.**

There is a tropical disease called beri-beri, which produces a chronic neuritis, leading to weakness and wasting of the muscles. There are also general œdema, fever, and anæmia, and the cause is probably bacterial.

It is remarkable that poisons circulating in the

blood and therefore reaching all the nerves of the body, nevertheless usually exercise a selective action, so that each poison nearly always picks out a particular nerve in every individual in whose blood it is circulating. For instance, lead nearly always affects the musculo-spiral nerve; alcohol most often the external popliteal; malaria the supra-orbital; and diphtheria the nerves of the palate.

Symptoms.—Lesions of **motor** nerves cause weakness (going on to paralysis), and wasting of the muscles. There may be spasms in the early stages. The reflexes are usually lost early, and the electrical reactions alter gradually, till even the reaction of degeneration may be obtained.

In **sensory** nerves lesions cause pain, usually in flashes, and sometimes the pain is accompanied by an eruption of herpes on the piece of skin supplied by the nerve. Eventually there may be anæsthesia.

In lesions of **mixed** nerves both pain and paresis occur.

DIPHThERITIC NEURITIS.

This is a parenchymatous neuritis, the nuclei of the primitive sheath swelling, and the myelin breaking up, before the increase of the neuroglia begins. The *symptoms* begin in the soft palate, which becomes paralyzed, so that food passes into the nose, and the voice becomes nasal. On inspection the patient cannot lift the soft palate voluntarily, and its surface is anæsthetic. Next the muscles of accommodation of the eye are usually affected, so that vision is impaired. The oculo-motor muscles may also be attacked, causing squinting, and afterwards any or all of the limbs may suffer. Even the bladder

may be paralyzed. The paralysis usually passes off slowly, the parts first affected recovering first. Massage and electricity, good feeding, and fresh air constitute the treatment.

THE CRANIAL NERVES.

The Olfactory Nerve.—The commonest lesion is *pressure by intracranial tumours*. The sense of smell becomes lost.

The Optic Nerve.—See Optic Neuritis, p. 8.

The Third Nerve.—This nerve supplies all the oculo-motor muscles except the external rectus and the superior oblique. It also supplies the sphincter of the pupil and the muscles of accommodation. Consequently, when it is destroyed the *symptoms* are external strabismus, from unopposed action of the external rectus, ptosis, and dilatation of the pupil. The commonest cause of this affection is the pressure of a tumour, very often a gunma, on the nerve at the base of the brain; but it may be due to diphtheria.

It must be remembered that the same symptoms may be produced by a lesion in the crus (see p. 25), but in these cases there is hemiplegia on the opposite side of the body to the affected eye.

The Fourth Nerve.—This supplies the superior oblique, so that if it be destroyed, the patient cannot turn the eye outward and downward.

Paralysis of the oculo-motor muscles is called *ophthalmoplegia externa*. If all the muscles be affected, the eyeball is motionless, and the patient has to move the head in order to change the field of vision. *Ophthalmoplegia interna* means paralysis of the iris and muscles of accommodation.

The Fifth Nerve.—This supplies the face with *sensation*, and its motor branches supply the temporals, masseters, and the pterygoid muscles—in fact, the muscles of mastication. Irritative lesions of these nerves are caused by cold, by malaria, by decaying teeth, or by pressure at the foramina. The *symptoms* are intense pain in one or other of the branches (trifacial neuralgia), and there may be tenderness at the supra-orbital notch, at the infra-orbital foramen, and at the mental foramen, *i.e.*, at the points at which the nerves emerge. In bad cases a mere breath of cold air may set up intolerable agony, and it may become impossible to wash the face. Hyperæmia of the skin supplied by the affected branches may occur during the paroxysms of pain. The drugs used for these cases are quinine, antipyrin, butyl chloral hydrate (gr. v.), exalgin (gr. iii.), opium, etc., and excision of a piece of the nerve, and even removal of the Gasserian ganglion have been performed. In mild cases aconite or belladonna liniment, laudanum, or the menthol cone, may be useful as local applications.

Destructive lesions of the fifth nerve cause anæsthesia of the whole of one side of the face (including the conjunctiva), the buccal mucous membranc, and the anterior two-thirds of the tongue, over which area taste is also lost. If the motor branch is also destroyed, there is paralysis of the muscles of mastication. Such cases are rare, and are usually due to tumours at the base of the brain. Sloughing of the conjunctiva and loss of the eye often follow the lesion.

The Sixth Nerve.—This supplies the external rectus, and its destruction causes internal squint.

The Seventh Nerve.—Paralysis of this nerve is called Bell's paralysis. The nerve may be injured (*a*) in its course from the medulla to the internal auditory meatus by tumours, chronic meningitis, etc.; (*b*) in its course through the temporal bone by necrosis following ear disease; and (*c*) on the face by cold, operations on the parotid gland, the pressure of forceps during delivery, etc.

The *symptoms* are paralysis of all the muscles of the face. The forehead cannot be wrinkled on the affected side, the eye cannot be closed, so that the conjunctiva may ulcerate, the tears overflow (from paralysis of the tensor tarsi), the fold by the nose is smoothed out, the lips cannot be tightly closed, and the corner of the mouth cannot be drawn out. The food tends to collect between the teeth and the cheek.

It used to be taught that if the nerve was injured in the temporal bone above the place where it is joined by the petrosal branch to the Vidian, paralysis of the soft palate occurred; but it is now considered that the soft palate is not affected in lesions of any part of the seventh nerve. Loss of taste in the anterior part of the tongue is, however, sometimes observed.

If the lesion be due to cold, as is very common, the muscles recover in a few weeks. They lose their reaction to faradism for a time, but do not give the reaction of degeneration. In necrosis of the temporal bone, however, the nerve is quite destroyed, so that the muscles waste and give the reaction of degeneration.

It will be remembered that the lower parts of the face become paralyzed when the lowest nucleus (see p. 67) of the seventh nerve is sclerosed in bulbar

paralysis. This can be distinguished from paralysis due to lesions of the nerve itself by the gradual onset, by the simultaneous paresis of the tongue from sclerosis of the hypo-glossal nucleus, and by the escape of the orbicularis palpebrarum for a long period or for ever. In lesions of the nerve all parts of the face are equally and simultaneously affected.

Further, in lesions of the facial tract above the nuclei of the seventh nerve the face is paralyzed. But the orbicularis palpebrarum escapes, being subject to Broadbent's law, and there is also hemiplegia. The hemiplegia is on the same side of the body as the facial paralysis if the lesion be above the pons, but on the opposite side if it be in the pons (see p. 25).

Auditory Nerve.—Lesions in this nerve cause deafness, even to the tuning-fork.

The lesions of the other cranial nerves are practically those of bulbar paralysis (see p. 66). Some cases of *torticollis*, however, are due to lesions of the spinal accessory nerve. The lesion is often congenital and of an irritative character, so that the sterno-mastoid is the seat of spasm, which may be continuous (tonic) or intermittent (clonic).

LESIONS OF CERTAIN SPINAL NERVES.

The *serratus magnus* is sometimes paralyzed from lesions of its nerve. The *symptoms* are inability to raise the arm higher than the shoulder, and projection of the lower angle of the scapula. If the *rhomboids* are also paralyzed, the scapula becomes lower and further removed from the vertebræ than normal.

The *intercostal nerves* are often the seat of neuritis. The *symptoms* are intercostal neuralgia,

and sometimes a herpetic eruption in the course of the nerves.

For lesions of the musculo-spiral and external popliteal nerves, see Lead-poisoning and Alcoholic Poisoning respectively.

The sciatic nerve may be affected by neuritis, or it may be pressed on by the gravid uterus or pelvic tumours. The result is sciatica, the symptom of which is pain in the course of the nerve, which is aggravated by sudden flexion of the thigh on the trunk. For the *treatment of sciatica*, see that of Facial Neuralgia, p. 89. In addition, blisters and the actual cautery have been used as counter-irritants to the skin over the nerve, and acupuncture of the nerve has been tried.

LEAD-POISONING.

Lead-poisoning is common among painters, plumbers, and all who work with white lead. In other persons it may occur from inhaling fresh paint or drinking water drawn from leaden cisterns, etc.

The possible effects of lead-poisoning are :

1. **Intestinal Colic.**—This consists in violent pain in the abdomen without any tenderness. Pressure may, indeed, relieve the pain. The patient feels as if he would be relieved by passing a motion, but his attempts to do so are ineffectual. There may be shivering, vomiting, and even cramps in the muscles. The abdomen is retracted, and there is absolute constipation.

2. **Peripheral Neuritis (Lead Palsy).**—The musculo-spiral is the nerve nearly always selected. Pains in the fingers and the arm result from the irritation

of the sensory fibres, and paralysis of the extensor muscles, leading to dropping of the wrist, follows destruction of the motor fibres. The supinator longus is affected late in the disease.

The other effects are : (3) **A blue line on the gums**, at their junction with the teeth. This is due to a deposit of sulphide of lead. (4) **Chronic nephritis** ; (5) **Gout** ; (6) **Defects of vision**, with sometimes optic neuritis.

Treatment.—To get the lead out of the system iodide of potash must be given. The colic should be treated by belladonna or opium combined with sulphate of magnesia.

ALCOHOLIC POISONING.

The possible pathological effects of alcohol are :

1. **Intoxication.**—This begins with a stage of exhilaration, followed by a stage of inco-ordination, in which the muscles of speech and of progression are most affected. Then the stage of depression comes on, often with vomiting, and the heart's action may be dangerously weak. In many cases deep coma occurs, with a flushed face and stertorous breathing. The pupils are dilated, but equal, and, unless the depression be very great, the pulse may be full and bounding.

Treatment.—The stomach-pump should be used, and, if the heart be weak, strychnia should be injected hypodermically.

2. **Subacute Gastritis.**—This occurs in the morning after a debauch. There is loathing of food, nausea, vomiting, headache, and a foul tongue. Nux vomica and carminatives, like ginger, may be given in effervescing mixtures. Further results follow if the alcoholism becomes chronic.

3. **Chronic Gastritis.**—The stomach is constantly bathed with mucus, and may even become fibrous from interstitial gastritis. The *symptoms* are habitual nausea and anorexia, especially in the morning. Bismuth is useful in these cases, and, if a stimulant be necessary, nux vomica and ammonia may be given.

4. **Peripheral Neuritis.**—As the external popliteal is the nerve most often affected, this disease has been called alcoholic paraplegia. The peronei and muscles on the front of the leg become weak and wasted, until finally the patient cannot walk. There is often pain in the legs from irritation of the sensory fibres, and the knee-jerk is lost early. Other parts besides the legs may, of course, be affected. Massage and abstinence from alcoholism will effect a cure in recent cases.

5. **Delirium Tremens.**—In this condition the patient is delirious, and in particular suffers from hallucinations. For instance, he may imagine that he sees snakes, or beetles, or rats, etc. He is often terrified, and tries to escape from imaginary enemies, or he may commit suicide in order to escape from his terrors. At the same time his muscles are weak and tremulous, but he is often strong enough to be very violent. Attacks of delirium tremens are usually preceded by drinking bouts, during which no food has been taken for days, and sleep has been entirely absent. Hence the *treatment* consists in giving food, forcibly if necessary, and in procuring sleep by large doses of chloral and bromide, and, at the worst, opium. Both the food and the hypnotics may be given by enemata, and hyoscine hydrobromate ($\frac{1}{100}$ grain) is very useful, as it can be given hypodermically. Physical re-

straint may be necessary, and the promises of the patient to be quiet must never be trusted.

It is important to distinguish delirium tremens from the delirium of the same kind which occurs when pneumonia attacks a habitual drunkard, for in the latter case the hypnotics are dangerous, and stimulants may be necessary.

The other effects of alcoholism are: (6) **Cirrhosis of the liver**; (7) **Chronic nephritis**; (8) **Chronic endarteritis and endocarditis**; (9) **Chronic meningitis**, which is usually found after death from delirium tremens; (10) **Sclerosis**; (11) **Amenorrhœa**; (12) **Fatty degeneration of the heart and liver**; (13) **Epilepsy**.

Dipsomania is the name given to the morbid craving for alcohol. The only means of curing dipsomania is to insist on *total abstinence*, since it is found that the slightest taste of a stimulant will revive the craving.

MERCURIAL POISONING

The possible effects are :

1. **Inflammation of the Gums.**—The gums become red, swollen, spongy, tender, and bleed readily, and there is a metallic taste in the mouth. The flow of saliva is increased, and the breath is offensive. In bad cases the gums may slough and the teeth fall out. As regards *treatment*, the mouth should be washed out with acid lotions or with astringents and antiseptics.

2. **Gastro-enteritis**, which may be acute or chronic.

3. **Mercurial Tremors.**—These are associated with the other signs of mercurial poisoning. The tremors usually begin in the hands and arms, but

may eventually affect all the muscles except the eyeballs. Sometimes the movements are jerky rather than tremulous, and they may be continuous. They are always made worse by excitement. Sensory disturbances, such as numbness or formication, or pains, may be observed.

Iodide of potassium and tonics should be given in all cases of mercurial poisoning.

DISEASES OF THE EAR.

THE EXTERNAL EAR.

Boils are not uncommon in the external auditory meatus, and they cause extreme pain. When they burst, there is a purulent otorrhœa. A large poultice should be applied over the ear, warm boracic lotion should be frequently injected, and the boil should be lanced as early as possible.

Accumulation of Cerumen.—This causes deafness, which usually comes on slowly, but sometimes suddenly, although the wax has been in the meatus for a long time. The hardened cerumen can be seen by the speculum as a dark mass filling up the meatus. It may set up inflammation, and cause pain and otorrhœa.

Treatment.—Warm sweet-oil (ʒss.) with tinct. opii (℥v.), or the same quantity of glycerine and water (ʒii. ad ʒi.) should be poured into the affected ear night and morning for two days, the patient lying on the opposite side for a quarter of an hour each time, so as to allow the fluid to soak in. Then the ear should be syringed out with warm water to remove the softened wax.

THE MIDDLE EAR—THE TYMPANUM.

Catarrh.—This is often due to cold, and extends from the pharynx along the Eustachian tube to the

tympanum. There is *deafness*, because the Eustachian tube is obstructed, and the air cannot enter the tympanum freely, a free supply of air on each side of the membrana tympani being essential to good hearing.

The Eustachian tube may be obstructed at its pharyngeal orifice by enlarged tonsils or post-nasal *adenoids*. Adenoids are fungating soft red masses, which microscopically consist of proliferated gland-tissue and granulation tissue. They result from chronic catarrh. In some cases of Eustachian obstruction the pharynx is pale.

Symptoms.—The *deafness* may be so extreme that a watch can only be heard when it touches the ear. The tuning-fork, however, is heard well when it is applied to the forehead, and it is usually heard most loudly in the ear which is most deaf to outer sounds. Some of these patients hear conversation best when a noise is going on, *e.g.*, in a train. *Tinnitus*, or buzzing in the ears, is often present, even when there is no deafness. With the speculum the membrana can be seen to be *retracted*, which makes the handle of the malleus prominent. This retraction of the membrana is due to the scanty supply of air in the tympanum. If air be blown through the Eustachian tube into the tympanum, the membrana can be seen with the speculum to bulge forwards. Inspection of the throat may show disease there.

Treatment.—The throat must be treated by gargles or astringent applications. Enlarged tonsils and adenoids must be removed. Glycerine of borax (ʒi. to ʒii. of warm water) should be syringed through the nose, so as to reach the orifices of the Eustachian tubes. Inflation of the tympanum by

Valsalva's method or by Politzer's bag is useful, and catheterization of the Eustachian tube may be necessary.

Suppurative Otitis Media.—This is usually due to the extension of suppurative faucitis to the tympanum in scarlet fever, but sometimes in measles and the other fevers.

Symptoms.—There is *deafness* and *acute pain* in the ear, with often *high fever*, and even *delirium*. The membrana may be red and bulging, while the mastoid process may be tender (from pus in the mastoid cells), and the skin over it red and swollen. If left untreated, the membrana usually ruptures, and blood and pus are discharged.

These cases tend to become chronic, so that the hole in the membrana does not close, and there is a continual purulent otorrhœa. Further, *polypi* may grow from the mucous membrane of the tympanum, and project through the hole, and fill up the meatus externus. They are composed of granulation tissue, and may be covered by epithelium. In neglected cases the tympanum (with the ossicles) becomes quite destroyed, the temporal bone becomes necrosed, and the internal ear disorganized. There is now complete deafness, even to the tuning-fork placed on the forehead. The facial nerve also becomes destroyed, so that facial paralysis results, and acute inflammation of the periosteum and skin over the mastoid may occur. The otorrhœa is very offensive. Finally, the septic process may extend to the meninges, causing meningitis; or to the brain, causing abscess; or to the lateral sinus, causing a septic thrombus, and very likely general pyæmia, with infarctions in the lungs.

Treatment. — At first opium and poultices or

leeches should be ordered to relieve pain, and the membrana should be incised. After incision or rupture the tympanum should be syringed out (sometimes through the Eustachian tube) with antiseptic lotions. In old cases, after syringing, boracic acid in powder, or iodoform, can be blown into the drum. Polypi should be removed by a forceps or the fine-wire ecraseur. In acute stages an incision down to the mastoid bone, or trephining the mastoid cells, or any part of the cranium beneath which pus is supposed to lie, may be necessary.

Perforation of the Membrana Tympani. — This may be due to otitis, as described above; or to violent coughing, as in whooping-cough; or, from without, to loud explosions. In traumatic perforation there is often a little bleeding. If the hole be small and the ossicles intact, hearing may be good. Patients with perforated drums can blow air through the ears by shutting the nose and mouth, and forcibly expiring. If there be inflammation, it should be treated as above, and if not, the case should be left alone. If necessary, an artificial membrana can be worn.

Fibrosis. — Inflammation of the tympanum may be chronic from the first, so that much fibrous tissue forms beneath the mucous membrane. This fibrous thickening stops vibration, and causes great deafness. The membrana is white and opaque, instead of gray and translucent. It may be due to syphilis or follow catarrh.

THE INTERNAL EAR.

Lesions in this position produce deafness even to the tuning-fork placed on the forehead (see Ménière's Disease).

OTORRHOEA.

Causes.—Boils or the irritation of cerumen in the external meatus, or suppurative otitis media with perforation. The discharge may be offensive even when there is no necrosis of bone.

EARACHE.

Causes.— Boils or hardened cerumen in the meatus externus, otitis media, and neuralgia. In the last case iodine or a blister behind the ear, and hot poultices or hot bran in a bag, may be tried.

DISEASES OF THE SKIN.

CLASSIFICATION.

Catarrhal Inflammation : Eczema.

Erythemata: Simple erythema, Urticaria, Roseola. The rash of scarlet fever and other exanthemata, and that of belladonna-poisoning, are also erythemata.

Papular Eruptions : Lichen, Strophulus, Prurigo. In these, the papules never become vesicles or pustules. Scabies produces a papule, but it may become pustular. In variola and varicella the pock is at first a papule, and afterwards a vesicle, and in variola it ends as a pustule. Even in eczema a small papule occurs before the fluid is sufficiently abundant to be seen.

Vesicular Eruptions : Herpes, Pemphigus. In eczema a vesicle is formed at an early stage, and in variola and varicella the pock always becomes vesicular. Sudamina are not true vesicles, because the fluid under the epidermis is sweat.

Pustular Eruptions : Ecthyma, Boils. In these suppuration is the essential point, but suppuration often occurs in eczematous vesicles and acne nodules, and constantly in variolous pocks.

Squamous Eruptions : Psoriasis, Pityriasis.

Hypertrophies: Ichthyosis and Xeroderma, Scleroderma, Keloid, Elephantiasis, Molluscum Fibrosum, Warts (Verrucæ), Corns.

New Growths : Lupus (tubercle), Rodent Ulcer, Cancer.

Pigmentary Diseases : Leucoderma, Addison's disease, Freckles (ephelides). As a result of constant scratching, pigmentation may occur in prurigo and phtheiriasis. It may also occur during pregnancy.

Diseases of the Glands—The Sweat-Glands : Sudamina, Miliaria, Anidrosis, Hyperidrosis. **The Sebaceous Glands :** Seborrhœa, Acne, Molluscum Contagiosum.

Diseases of the Hairs : Alopecia areata, Tinea tonsurans, Sycosis, Favus. The last three are parasitic.

Parasitic Affections—Vegetable Parasites : Tinea tonsurans and circinata, Favus, Sycosis, Pityriasis versicolor. Some authorities think that alopecia areata is parasitic. **Animal Parasites :** Scabies, Phtheiriasis.

Many skin affections, such as herpes, urticaria, etc., appear to be due to nervous influence. Simple pruritus, *i.e.*, itching without any obvious cause, is considered to be a neurosis.

Hæmorrhages occur in the skin in purpura and other cachectic states. Many rashes are at first erythematous, but later on become hæmorrhagic (*e.g.*, the typhus rash).

It must be remembered that **Syphilis** may produce almost any variety of skin affection.

ECZEMA.

In *acute* eczema large numbers of small red papules rapidly appear on the hands, face, trunk, and limbs, chiefly on the flexor surfaces. They itch and burn intensely, and erythema may occur

between them. The papules soon become filled with fluid, and then burst, discharging a sero-purulent fluid. This fluid dries, and forms crusts. The affection tends to get well in a few weeks.

Subacute eczema is more common, and lasts longer. The process described above goes on, but more slowly, and the crusts may be very extensive. Sometimes large blebs filled with pus are formed, most often on the scalp (especially in children), but also on the trunk and limbs. This is called *eczema impetiginodes*, or *impetigo*. When the blebs burst, the pus dries to large crusts, which resemble syphilitic rupia; but on removing the crusts the skin is not raised as in rupia, and after healing there is no scar.

Eczema at the flexures of the joints is called *eczema intertrigo*. In *chronic eczema* there is less vesiculation, and therefore less discharge.

Causes.—The cause is unknown, but it may be due to gout. The micro-organisms of the skin aggravate the disease.

Treatment.—The crusts must be removed by softening them with warm water or poultices. Then ung. zinc., or ung. boracis, or salicylic cream must be applied to the raw surfaces. This process has to be repeated several times, because the crusts re-form, but each time to a less extent. Itching may be relieved by lead lotion or by an ointment containing potass. cyanid. (4 grains to an ounce of ung. cetacei).

Internally, saline aperients should be given at first, and then liq. arsenical., ℥iv. t. d. s. Chloral or opium may be necessary to procure sleep in acute cases. The diet should be chiefly farinaceous, and malt liquors and heavy wines should be avoided.

Soap should never be used, but the skin may be washed with oatmeal-water.

ERYTHEMATA.

These are diffuse red rashes, which disappear on pressure, and are only slightly raised.

1. **Simple Erythema.**—Patches of erythema often occur from unknown causes, perhaps from digestive disturbances, particularly in hot weather. If very transient, they are called erythema fugax. **Erythema Nodosum** consists of large patches of erythema (most often on the front of the legs), followed by rupture of the capillaries, and consequent hæmorrhage. The effused blood raises the patch, and gives it a purplish colour, which changes to blue and yellow, like a bruise. The patches are painful and very tender. They are associated with rheumatism.

2. **Roseola.**—This consists of an eruption of small pink, slightly raised patches. It may be very extensive, and is most common in summer. There may be slight fever. It is very like measles, but there is no coryza, and the fever is less.

3. **Urticaria** (Nettlerash).—This begins as diffuse red patches, which itch and sting intensely. Soon whitish soft projections like wheals appear on the red surface from localized œdema of the skin. Large surfaces of the body may be covered by the rash. Urticaria may be caused by eating shellfish or mushrooms, but in many cases the cause is unknown. It has been attributed to nervous influence. The rash is like that produced by stinging-nettles.

4. **Syphilis.**—The earliest rash of acquired syphilis is a roseola. It consists of small purplish patches,

which occur on the chest or abdomen, but may be universal.

Treatment.—All the erythemata except the syphilitic may be treated by aperients and salines. For erythema nodosum tonics are necessary, and the salicylates may be tried. If urticaria be due to errors of diet, an emetic may be given, and the itching may be allayed by lotions containing ammonia or prussic acid.

PAPULAR ERUPTIONS.

Papulæ are small, firm, more or less pointed projections, which in most cases are formed by swelling of the normal papillæ of the skin.

1. **Lichen.**—In this disease minute red papules occur in the skin, either in circumscribed groups or in large patches. They appear very suddenly, itch severely, and may even be accompanied by fever. They never form vesicles, and they disappear in a week or two, with perhaps some desquamation. The *prickly heat* of hot climates is called *lichen tropicus*. Saline aperients and sedative applications should be prescribed, as for urticaria.

2. **Strophulus.**—This occurs in children, generally during dentition. It consists in an eruption of small pink papules all over the body, which do not appear to itch much. The diet and the teeth must be attended to.

3. **Prurigo.**—This disease may begin in children (more often in males), and is attributed to nervous influence. Small, pale, discrete papules form on the extensor surface of the limbs and on the back. They itch intolerably, and the constant scratching leads to pigmentation and thickening of the skin,

and to the formation of blood-crusts at the top of the papules. The disease is practically incurable, but relief may be obtained by sedative lotions and ointments, or by keeping the patient in a bath.

VESICULAR ERUPTIONS.

A vesicle is a small rounded projection from the skin, formed by a collection of clear lymph which has pushed up the epidermis. Bullæ, or blebs, are large vesicles.

1. **Herpes.**—In this disease there is an eruption of vesicles. The following varieties are described :
 (a) *Herpes Zoster*, in which groups of vesicles like bunches of grapes form rapidly on the skin of the chest, at the points where the chief branches of the intercostal nerves supply the skin. In this way the groups of vesicles may encircle the chest like a girdle. The affection is called *shingles*. The name *zoster* is also sometimes given to herpes when it occurs in the limbs in the areas supplied by the branches of a cutaneous nerve. The formation of the vesicles is accompanied by neuralgic pain, the vesicles themselves itch and burn intensely, and the raw surfaces left after they burst are very painful. The eruption is due to irritation of the sensory nerves by neuritis or by the pressure of a tumour. The pressure of the uterus in the last week of pregnancy has been known to cause herpes along the course of the sciatic nerve. In pneumonia herpes often occurs on the face in the part supplied by some branch of the fifth nerve.

(b) *Herpes Labialis*.—The vesicles form on the lips. This often occurs in the course of a common cold or from gastric irritation.

(c) *Herpes Præputialis*.—The vesicles form on the prepuce.

(d) *Herpes Iris*.—In this disease the vesicles are not round, but more or less elongated, and they are curved one around the other.

Treatment.—Pain may be relieved by opium, and the vesicles protected by sedative ointments.

2. *Pemphigus*.—Large blebs, with red margins like blisters, form on the skin in varying numbers, and cause the same kind of pain as a blister. They may contain clear lymph or pus and blood. The disease is very obstinate, and when cured is liable to recur. In old people it may cause death from pain and exhaustion.

Treatment.—Opiates may be given to relieve pain, and arsenic and other tonics may be tried. Sedative applications are also necessary.

PUSTULAR ERUPTIONS.

A pustule is a small rounded projection from the skin, formed by a collection of pus which has pushed up the epidermis.

1. *Ecthyma*.—This is the name given to an eruption of simple pustules. It is most common on the back and lower limbs, and there may be some pain and itching. The general health should be treated, and locally mild antiseptic applications, such as boracic lotion, should be used. It may be necessary to apply iodoform to the site of the pustules after they have burst, or even the solid nitrate of silver.

2. *Boils*.—These are rather more than mere pustules, as the pus forms round a core, *i.e.*, a collection of cells which have undergone necrosis. There is considerable redness round a boil, and

there may be great pain. Sulphide of calcium ($\frac{1}{6}$ grain) in a pill may be given every four hours, and early incision is advisable. As the true skin is involved, a scar is always left.

3. The conical-shaped crusts of inspissated pus which are called *rupia* usually result from the rupture of syphilitic pustules.

SQUAMOUS ERUPTIONS.

These are characterized by the formation of dry scales on the top of a red raised base.

1. *Psoriasis*.—In this disease a red, slightly elevated patch of varying size, with a flat top, first forms on the skin. Very soon the top becomes covered by a white glistening scale, composed of the superficial epidermal cells which are glued together. A large patch is covered by many scales. The scales come off in a few days, but are immediately renewed. Sometimes the formation of the patches is accompanied by much itching. The cause of the disease is not known, but it often occurs in gouty persons or their children.

Varieties—(a) *Psoriasis Vulgaris*.—In this form large patches occur on the extensor surfaces of the limbs or body, especially on the back of the elbows and the front of the knees. A few patches occur on the flexor surfaces, and the forehead and scalp are often affected.

(b) *Psoriasis Guttata*.—In this form the patches are small, few, and separated widely from each other like scattered drops.

(c) *Syphilitic Psoriasis*.—In this form the base of the patches is coppery rather than red, and the scales dirty-looking rather than silvery. The flexor

surfaces are chiefly affected, and itching is said not to occur. In *palmar syphilitic psoriasis* there is no regular eruption, but the palms of the hands become dry and extraordinarily cracked and fissured.

The Treatment of non-syphilitic psoriasis consists in applying ung. acid. chrysophanic. or ung. picis liquid. locally, and giving arsenic internally.

2. *Pityriasis*.—In this affection fine branny scales form on the skin, especially on the scalp. There is no thickening of the skin, but in rare cases the skin is bright red, and the affection is then called *pityriasis rubra*. In some cases there is much itching. Tonics should be given, and zinc ointment, lead lotion, liquor carbonis detergens, etc., applied.

HYPERTROPHIES OF THE SKIN.

1. *Ichthyosis*.—This affection is nearly always congenital, and appears shortly after birth. The skin becomes thickened, hard, and dry, with irregular flattened projections of the epidermis, resembling the skin of a crocodile or that of a fish (whence the name *ichthyosis*). It may affect the whole skin, but is most marked on the extensor surfaces. The hair and nails are brittle, and the patients are very sensitive to cold; but there is no pain or itching. *Xeroderma* is a lesser form of the affection. The disease is incurable, but inunction with fatty materials, like lanolin, is recommended after the skin has been cleaned with alkaline lotions.

2. *Scleroderma*.—In this affection the skin becomes thick, hard, and rigid; but it is not rough

and scaly as in ichthyosis. It is drawn tightly over the subjacent parts, making the joints immobile, and converting the face into a mask. It is not reddened. When the disease occurs in localized patches or bands, it is called *morphæa*. Fattyunctions and tonics are recommended as treatment.

3. **Keloid** consists in a localized hypertrophy of the true skin. The affected part looks like a raised, pinkish scar, often with claw-like off-shoots from the central mass. It is a very rare disease, and may be due to the hypertrophy of the scar tissue after a wound.

4. **Elephantiasis** is a hypertrophy of the true skin and epidermis, and of the subcutaneous connective tissue as well. It is usually due to the blocking of the main lymphatics of the leg by the *Filaria sanguinis hominis*, but it may result from lymphangitis. This causes the lymph to accumulate in the leg, and so produces a semi-solid œdema, which is followed by the hypertrophy.

5. **Mollusum Fibrosum**.—In this disease a varying number of more or less pedunculated tumours grow from the skin in pendulous masses. They consist of hypertrophied skin papillæ, *i.e.*, of connective tissue, fat, and bloodvessels, and they may be as large as a turkey's egg, or even larger.

NEW GROWTHS.

1. **Lupus**.—This is a tuberculosis of the skin. The tubercles form little nodules in the skin (which is usually inflamed), and, as in other positions, they undergo caseation. In some cases they spread with difficulty, and rather tend to become invaded by granulation tissue, and so to become healed.

This is called *lupus non-exedens*. In other cases—*lupus exedens*—they soften rapidly, and lead to extensive ulcerations. Lupus is most common on the face, and, in old cases, disfiguring scarring may be seen where the tubercle has healed, and ulcers where it has recently softened, while at the periphery fresh nodules may be developing.

Treatment.—The nodules may be curetted away, and skin-grafts implanted on the raw surface, or corrosives, such as the acid nitrate of mercury or chloride of zinc, may be applied to the tubercles. Cod-liver-oil and tonics should be prescribed.

2. **Rodent ulcer** begins as a slowly-developing nodule, which slowly ulcerates. The ulcer becomes excavated, and has thick raised edges. The nodule is a morbid growth like epithelioma, but the cells are smaller than squamous epithelium, and cell-nests rarely occur. The whole ulcer should be excised.

PIGMENTARY DISEASES.

1. **Leucoderma.**—In this disease patches of skin of various sizes become quite white from the removal of the natural pigment. The skin around is usually darker than normal, and the line of demarcation between the white and dark patches is sharply defined. This distinguishes leucoderma from Addison's disease, in which the pigmented parts shade off gradually into the healthy skin. The hairs in the affected parts become white. Leucoderma is most common in mulattos.

THE SWEAT-GLANDS.

Sudamina.—During profuse sweating the ducts of the sweat-glands may become blocked, and

the sweat then distends the epidermis, forming multitudes of small shining pseudo-vesicles, filled with clear fluid. These are called sudamina, and they are common in rheumatic fever. If a ring of redness forms around them they are called *miliaria*.

Excessive sweating is called *hyperidrosis*, and deficient sweating *anidrosis*.

THE SEBACEOUS GLANDS.

1. *Seborrhœa*.—In this disease there is excessive secretion of sebum, so that the parts where sebaceous glands are abundant, such as the scalp, become covered with small fatty scales. As treatment, ointments containing astringents, such as tannin, or antiseptics, such as sulphur, have been recommended.

2. *Acne*.—This consists in the accumulation of sebum within the sebaceous glands from blocking of the ducts. It occurs most often on the face, particularly in youths at the time the beard is appearing; but it is also common on the back. The distended glands project slightly, and there is often a black spot of dirt at the apex of the projection. These white nodules with the black spots are called *comedones*, and can be cured temporarily by squeezing out the sebum. More often the gland inflames, so that it becomes a hard red papule, which is very indolent, but may suppurate, and even leave a scar.

Acne rosacea is a variety in which the acne spots are surrounded by areas of inflamed skin. It is very chronic, and occurs in middle-aged people.

It may be due to alcoholism, or to gastric or uterine irritation.

Treatment.—Saline aperients should be ordered, and the stomach or uterine affection should be treated. Locally, ung. sulph. odid. is recommended.

3. Molluscum Contagiosum.—In this disease the glands become distended with very soft sebum, and project from the skin as yellowish nodules, the size of a pea or larger. There are always many nodules, and they are contagious. The nodules should be incised, their contents removed, and the base cauterized.

DISEASES OF THE HAIRS.

Alopecia Areata.—In this disease the hairs atrophy over a localized portion of the scalp, which becomes bald and smooth. The bald patches are circular, but may be distinguished from ringworm by the fact that no broken hairs, nor epidermal scales, nor signs of inflammation are present. The disease is thought by some to be of nervous origin, and by others to be parasitic. The treatment consists in applying acetum cantharides or ung. chrysarobin. to the bald patch.

The other affections of the hairs are treated with the parasites.

THE VEGETABLE PARASITES.

1. Ringworm.—This disease is due to a fungus called the *Tricophyton tonsurans*, which consists of rounded or oval spores, and a mycelium composed of thickish short threads. The fungus enters the hairs, and developes among the fibres of which the hair is composed. At particular points it may give

a beaded appearance to the hair, and at this point the hair breaks across, the broken ends being very irregular. The parasite is also found at the roots of the hairs. The varieties of ringworm are:

(a) *Tinea tonsurans*, or ringworm of the scalp. In this, the destruction of the hairs causes a circular bald patch, in which the broken hairs may be seen. As the parasite irritates the skin, redness and vesiculation, and subsequently the presence of epidermal scales, may be observed.

(b) *Tinea circinata*, or ringworm of the body. In this the parasites attack the fine hairs and epidermal cells, and cause the formation of patches, surrounded by curved lines of inflammation.

Treatment.—Iodine liniment or acetum cantharides must be painted on to the patches. The scalp must be shaved, and epilation may be necessary.

2. **Sycosis** consists in the formation of papules or pustules (like those of acne) in the hair follicles of the beard, etc. The skin around is inflamed, and crusts of inspissated pus may be present. The hairs fall out. The disease is now thought to be due to staphylococci or to a special bacillus. As treatment, epilation and the application of oleate of mercury or sulphur ointment are recommended.

3. **Pityriasis versicolor** is due to a fungus called the *Microsporon furfur*, which has a branched mycelium and large spores arranged in clusters. These infest the epidermis and fine hairs, most often on the chest, and cause pinkish-brown lines or bands arranged in loops or curves, and covered with soft scales. It is easily cured by applying iodine liniment.

4. **Favus** is due to a fungus called the *Achorion*

Schænleinii, which consists of spores and long branching threads of mycelium. It attacks the hair follicles and epidermis. The result is the formation of small round sulphur-coloured crusts, which are concave at the top, and are perforated by a few broken hairs. On removing the crusts the skin is also seen to be slightly hollowed out. The crusts are much more common on the scalp. They are said to smell of mice, and it is thought that the disease may be caught from mice, or rabbits, etc., by uncleanly persons. The treatment is the same as that of ringworm, but the crusts must be carefully removed.

All the diseases caused by vegetable parasites are contagious. The name *tinea*, which means a worm, is often applied loosely to them, *e.g.*, favus is often called *Tinea favosa*.

THE ANIMAL PARASITES.

1. *Pediculi* (Lice).—There are three varieties: *pediculi capitis*, *pediculi pubis*, and *pediculi corporis*. The last lives in the clothing. They all cause intense itching, and may lead to eruptions. In chronic cases the skin becomes thick, pigmented, and papular from much scratching. This condition of skin is called *phtheiriasis*. On the scalp and pubes the eggs (nits) are firmly attached to the hairs.

Treatment.—Ung. hydrarg. ammon. should be rubbed in, as it kills both the lice and the eggs. It may be necessary to shave the parts, and infected clothing should be destroyed.

2. *Scabies* (the Itch).—This disease is due to the *Acarus scabiei*, which is a minute insect.

Under the microscope it looks like a cockroach, having a flattened body, with four short legs anteriorly and four smaller ones posteriorly. A number of fine spines project from the sides. The female is about a quarter of a line in length, the male about two-thirds of that size. The male is migratory, and is seldom found, but the female burrows into the epidermis, where she causes the formation of a small papule, which, with the burrow leading to it, may be quite visible. Here she lays her eggs. The papules are always situated on flexor surfaces, especially between the fingers and toes, and they itch intensely. They may suppurate. The disease is easily cured by the free use of sulphur ointment for two or three days.

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